THE
LARYNGOSCOPE.

VOL. L APRIL, 1940. NO. 4

PHYSIOLOGY OF THE LARYNX. A RESUME AND DISCUSSION OF THE LITERATURE FOR 1939.

DR. JOEL J. PRESSMAN, Los Angeles.

Negus in his text[21] has brilliantly described the primary function and evolutionary background of the larynx as a sphincter mechanism, the chief purpose of which is to prevent the invasion of foreign matter into the lower respiratory passages. We seldom think of the larynx in this light, but rather primarily as the organ of voice or sound production. Its importance as a sphincter valve is frequently minimized or entirely overlooked. For this reason I have chosen an article by an internist, I. L. Appelbaum,[1] with which to begin this year's abbreviated resumé and discussion of current literature dealing with the physiology of the larynx.

Appelbaum has studied the activity of reflexes of the upper respiratory tract, especially those of the larynx and pharynx, under a variety of conditions, and in various pulmonary diseases. The pharyngeal reflexes were observed by using a wooden tongue depressor and noting the degree of gagging. Laryngeal reflexes were tested by touching the laryngeal surface of the epiglottis with a curved applicator and noting, by mirror observation, the laryngeal response. [Unfortunately no very accurate level of normal response can be established or described as a basis for comparison, but nevertheless a crude general impression of the reactions can be obtained and I do not believe the author intends to imply more than this. The same objection may be raised on the basis of there being no established normal to a description of knee-jerks as

Editor's Note: This ms. received in Laryngoscope Office and accepted for publication, March 30, 1940.

"hyper-" or "hypoactive," but within reasonable limits we have come to recognize such deviations.—J. P.] In 75 per cent of all cases described as having diminished upper respiratory reflexes, there was a corresponding diminution in the general sensitivity of the nervous system as determined by testing other reflexes.

In normal adults about 7 per cent presented depressed laryngeal reflexes, and 12 per cent depressed pharyngeal reflexes. This number was increased to 30 per cent and 33 per cent in acute alcoholics, and in patients under sedative drugs. In keeping with this observation, when lipiodol was dropped into the mouths of adult persons normally asleep, 16 per cent aspirated the oil into the lungs. (Only 4 per cent of children aspirated the oil.) In alcoholic states, the percentage increased to 56 per cent, and under sedatives to 40 per cent. The author concluded that alcohol and sedation (and to a lesser extent normal sleep) depressed the laryngeal and pharyngeal reflexes and tended to render the protective laryngeal sphincter action, and cough reflex nonactive. Appelbaum then studied 100 cases of lung abscess and noted that 85 per cent suffered acute or chronic upper respiratory infections. Of these 86 cases, 85 per cent had depressed laryngeal or pharyngeal reflexes (and in most of these a combination of both) due to a variety of causes, such as alcoholism sedation, epilepsy, etc., including 10 with a generally depressed reflex state. The author, therefore, believed that he had found in 85 per cent of a large series, an etiologic basis for lung abscess, this being the coexistence of diminished upper respiratory reflexes and a source of upper respiratory infection, usually pyorrhea. In a very much smaller percentage of cases the syndrome was found to be present in a series of bronchiectatics.

The obvious conclusions drawn were to stress the importance of the laryngeal reflex as the "watch dog of the lungs" and to caution against the use of narcotics in all upper respiratory infections.

[It is not my purpose to take issue in any way with the contents of this article. I feel that the data is accurate and the conclusions justified. Chevalier Jackson has for years laid stress upon these same physiopathologic conceptions, and they can be taken almost for granted. Nevertheless, one clinical

observation in my experience bears discussion. Professional singers, especially those who have been highly trained, almost invariably present no gag reflex and very little by way of a laryngeal reflex. It is possible to insert a laryngeal mirror into the pharynx of such a person or to paint their throats without producing gagging, and to insert oils into the larynx without producing cough or stimulating the sphincter mechanism into action. I have observed this phenomenon so often that in my own mind there is no doubt of the accuracy of the observation. If this is true, then professional singers as a group should be more susceptible to lung abscesses than the general cross-section of population, since they are no less susceptible to upper respiratory infections and almost universally present the necessary diminished upper respiratory reflexes to complete the syndrome; however, I have not noticed any such increase in the incidence of lung abscesses amongst singers.—J. P.]

B. T. King,[3] in discussing the possible regeneration of fibres of the recurrent laryngeal nerve following resuturing for paralysis, points out that the recurrent laryngeal nerve carries adductor and abductor fibres in the same sheath. This makes nerve suture almost impossible since the proper "hook-up" of neurons would be very difficult to establish. He also indicates that by the time regeneration occurred, atrophy of the muscles would have taken place to the point of permanent contraction.

Chevalier and C. L. Jackson[4] have contributed a paper so important to the welfare of the professional vocalist that it should be studied and its general principles memorized by every laryngologist. It should be brought to the attention of everyone to whom a good voice is of value. This contribution is indeed a classic, outlining in dramatic and convincing fashion a great many points of vital importance in daily office practice. Because of its importance I am quoting freely from the paper and presenting its contents rather fully, as follows:

A pleasant voice may have developed naturally, and others have a naturally disagreeable voice. Both can be improved by proper training in early life. This needs no special course of study other than good example and a few words of admonition.

Lack of clear, distinct enunciation is one of the chief reasons for vocal abuse and has a distinct bearing on the larynx. Most people when not understood "force" the voice instead of repeating clearly rather than more forcibly.

Most great vocalists, if they continue long enough, lose their vocal power so that the voice weakens and roughens and the quality is lost.

The singing of the professional vocalist is at best an unnatural use of the larynx. The singer puts a great strain on the larynx in extending the limits, especially the upper limits, of the scale and by unusually prolonged and forceful use. This is often done with reckless disregard of all care, and in those with this priceless gift, a fine voice is often maltreated, a comparison paralleled only by hitting a Stradivarius with a hammer. It is stressed that professional vocalists of all professions have the very bad habit of talking between times, so that the larynx gets no rest. Other abuses are talking too loudly and talking in noisy places. Our civilization is becoming more and more noisy and this is injurious not only to the nervous system but to the larynx as well, forcing us to shout.

[The Jacksons list many causes of vocal abuse in addition to the above. Alcohol in any quantity is injurious to the larynx. This dilates the capillaries of the mucosa and anyone with hopes for a vocal career should be a total abstainer. With the vocal cords in a swollen state, the alcohol, in addition to the swelling, almost always causes an added tendency, in even mild alcoholic states, to talk more loudly, which, considering the swollen state of the cords, makes this abuse doubly harmful.—J. P.] Even minimum amounts of alcohol (a single drink) are enough to produce this state of engorgement.

Tobacco smoke is injurious to the larynx. This is due to the presence of an oil produced by the destructive distillation of the tobacco when smoked.

Each severe and prolonged use of the voice should be followed by a period of silence. Many a professional career has been wrecked by the use of a voice during an acute upper respiratory infection.

Speaking or singing outdoors is especially harmful. The vocalist does not get reflected back to him the sensation that

he is "filling the hall." Consequently, more and more force is put into the voice until laryngeal muscles are strained or capillary blood vessels on the cords are ruptured. This may lead to vocal nodes or hemangiomata. Caution in use of the voice outdoors is, therefore, especially stressed. A dangerous triad is alcohol, tobacco and shouting outdoors. [Nothing much can be added to this. It occurs to me that a word of warning might be advanced in regard to professional vocal training of young children. Almost every parent whose young offspring presents a voice of promise rushes to a vocal teacher in order that training might begin as early as possible and a professional career begun without delay. The delicate, undeveloped vocal cords of a young child or adolescent cannot withstand the rigors of long hours of practice, and the vigorous use of the larynx that vocal training and professional usage requires. This is especially true when the voice is changing and the cords are particularly apt to be congested and increasingly susceptible to injury. Vocal efforts of the immature larynx should be decidedly limited. No extremes of range are to be attempted, volume should be greatly reduced and hours of usage limited to a minimum. The force of the expiratory blast of the lung of a growing child is ordinarily greater in proportion than the state of development of the vocal cords, so that forceful, prolonged use of the voice results in proportionately greater trauma to the vocal cords. If a career as a vocalist seems in the offing, I know of no better way to ruin it than too early and too enthusiastic training.—J. P.]

In speaking of the immature larynx, Jannulis[5] makes a number of interesting observations. In some males, breaking of the voice is observed at puberty. There appears at puberty an abrupt interchange of "chest" and "head" voice, which may persist and lead to a prolonged mutation. A still more obnoxious feature is persistent falsetto. This falsetto is originated also by an abnormal action of the cricothyroid muscle. In the treatment of persistent falsetto, he proceeds by interrupting the upper laryngeal nerve by local anesthesia. By this method of putting out of function the superior laryngeal nerve and the muscles it supplies, the patient speaks in a deeper voice. If once the patient speaks in a normal voice under anesthesia this will continue after the anesthesia has worn off. After this treatment patients develop a subjective anesthetic differentiation of tone and are annoyed listening to their own falsettos and to that of other patients.

This disorder has a depressing effect on patients since they are hampered in their professional and social occupations.

In toxic laryngodynia after influenza without visible inflammatory changes in cords, the authors were able by anesthetizing the superior laryngeal nerve to free patients of pain. There resulted a secondary deepening of the voice, from which they conclude the cricothyroid muscle was rendered functionless. [In our bronchoscopic clinic we have for years routinely anesthetized the superior laryngeal nerve prior to each bronchoscopy. Not once, however, have we noticed any very appreciable lowering of the voice.—J. P.]

In normal dogs there resulted a roughening of the bark when the superior laryngeal nerve was anesthetized without trauma to larynx.

The eunochoid voice demonstrates a deficiency in the development of the larynx. In these voices the chest register is absent, and the head voice functions a little, but timbre is missing. Paralysis of the superior laryngeal nerve in eunochs results in a deepening of the voice. In castrated dogs there was observed a roughening of the bark when the nerve was rendered functionless.

The authors further state that interruption of the passage of impulses through the superior laryngeal nerve, even in normals, results in slight hoarseness due to the paralysis of the cricothyroidens muscles.

Trendelenburg,[6] by measuring the width of the rima glottidis and by observing "air-sound curves" (oscillograph tracings?), has been able to demonstrate that when the rima is more widely opened the vibrations of the vocal cords are of greater amplitude than when the rima is closed. [This corresponds to observations reported later in this resumé in the paragraph dealing with the laryngeal motion pictures of the Bell Telephone Co.[14] In this latter discussion it is pointed out that when the rima is relatively more closed the pitch of the resulting tone is higher, and variation in the width of the chink of the glottis during vibratory movements of the cords is then very slight as compared to the amplitude of cord movements during the production of lower tones, when the chink is wider.—J. P.] Trendelenburg also speaks of the effect on overtones of changes in the shape of the resonating chambers. For example, in changing from one vowel to another the shape of the resonating chambers about the mouth cavity varies considerably, which in turn causes a variation in over-

tones and consequent changes in the quality of the sound produced.

Mr. William A. C. Zerffi[7] writes on functional vocal disabilities and quotes Robert A. Ridpath to the effect that "there are few laryngologists who have more than a meagre knowledge of the art of vocal training, and there are fewer vocal teachers who have any knowledge of the anatomy, physiology or the phenomenon of voice production." Into which category he falls, however, Zerffi fails to state. This author convinces himself that the treatment or rather the "no treatment" of vocal nodes, "irritations of the vocal processes" and contact ulcers, to mention only a few, is considerably beyond the capabilities of the laryngologist. He points out that many years of study of the subject have convinced him that only a very radical change in the handling of such situations can be expected to yield a satisfactory solution. The studies involved, and by which he was convinced, are not further mentioned but apparently consist in placing the finger "above the larynx in the angle of the chin," whereupon swallowing reveals "the co-operation of the external muscles. If the finger be returned to the original position and an attempt made to sing or speak, any muscular movement akin to that induced by swallowing reveals an unnecessary involvement of the external muscles. That is to say, the vocal cords are being brought together with greater force than their true muscles can develop and, consequently, there is danger of irritation of the free edges of the cords." [I tried this and it doesn't seem to work, but perhaps my impressions may change after the necessary years of study with the finger maintained in the approved position.—J. P.] This in no way, however, minimizes the value of the author's observations because he himself says that the recognition of such faults of voice production involves a deep and lengthy study, such as would "be outside the activities of the laryngologist"; however, Mr. Zerffi points out that the detection of this phenomenon can be a relatively simple matter, as described above, in which case I presume it would not be outside the activities of the laryngologist. What the nature of the radical change in the handling of the aforementioned situations might be, Mr. Zerffi does not indicate. With laryngologists knowing nothing about vocal training and vocal teachers having no knowledge of laryngeal anatomy, physiology or the phenomenon of voice production, there seems to

be no hope at the moment. The solution perhaps lies in the author's theory that the improper use of the muscles of deglutition leads to a too forceful closure of the vocal cords. That this takes place is proven by the fact that infants "produce sounds of far greater volume than might reasonably be expected," although he does not point out how much in the way of sound we have a right to expect from a given infant. This discrepancy, he states, is due to the fact that the "extremely weak" laryngeal muscles of the infant are aided and abetted by the more powerful deglutory muscles, and the partial laryngeal closure thus produced enables the infant to produce so great a volume of sound. [Actually and in fact, the volume of the tone produced represents several combined factors, including the force of air pressure from the lungs below and the nature of the resonating chambers above and below the larynx. No evidence, however, is introduced to prove the author's contention that the deglutory muscles are more powerful than the internal laryngeal tensors or, for that matter, that they have anything to do with vocal cord movements.—J. P.]

The author suggests that a careful and thorough analysis of the action of the internal or true laryngeal muscles will reveal the fact that these muscles are literally incapable of acting in such a manner as would enable the vocal cords to be forced together. [This is contrary to all accepted views and could be given credence only if we were to entirely disregard all available anatomical and physiological evidence, as well as that furnished by a study of the distribution of the laryngeal nerves and the results which follow their paralyses both clinically and experimentally.—J.P.]

Hartman and Wullstein[8] report studies carried out by means of shining a powerful point of light through the vocal cords from below, and registering, upon a photoelectric cell placed above the larynx, the amount of light passing through the vocal cords in different phases of phonation. The method is an improvement over that originally described by Trendelenburg and Wullstein. The experiment was carried out upon untreated vocal cords of calves and upon those treated with dyes to partially absorb the light, but not enough dyes were used to affect the vibratory movements of the cords. This experiment is intended to demonstrate the changes in "mass" and "mass-movements" of the vocal cords during the

phonatory cycle. Obviously, the thicker the body of the vocal cord happens to be at the moment the smaller will be the quantity of light shining through.

The vocal cords are approximated by a suture placed through the vocal processes, and air from below forced through under pressure, simulating the normal expiratory blast as it occurs during phonation. From these experiments the author was able to conclude: *1.* that the opening phase of vocal cord movements takes place more gradually than the closing phase; *2.* that the cords are very markedly thinned and become progressively thinner at the very beginning of the opening phase, so that large quantities of light shine through even the dyed cords. This thinning is greater than at rest. The thinning decreases as opening progresses until at the end of the opening phase the mass of the cords re-establishes itself to that usually present in the resting stage. The closing phase then begins and the cords become thicker (very little light shines through). In human cadaver larynges, the entire cycle of opening and closure is accompanied by the same relative changes in mass, except that the thickness during any part of the cycle is never equal to that seen during rest. In other words, during all phases of the opening and closing activities the cords are at least partially thinned as compared to rest. The thinning of the vocal cords in the human, just prior to the opening of the cords, takes place more rapidly than in the calf so that the vocal cords of the human are more easily blown apart. The human larynx responds to smaller blasts of air than the calf and is, therefore, more sensitive to these stimuli. *3.* Higher frequencies are accompanied by a shortening of the opening phase, including the preceding vocal lip thinning, while the closing phase possesses usually a constant duration (regardless of pitch?). *4.* In higher frequencies the rapidity of the vibration is such that sufficient time does not elapse during the closing phase for the cords to become as thick as they do at lower frequencies or at rest, so that some increased amount of light shows the vocal lips during all phases of phonation even in the dyed larynx. [I would assume from this that the cords are thinned more during comparable stages of the production of high tones than of low tones, but cannot agree that this is entirely due to the time element involved. It would seem more likely that the greater tension of the vocal cords, and their elonga-

tion, during the production of higher tones would naturally tend to thin them, even without any consideration of the factor of time.—J. P.] 5. These thickenings and thinnings of the vocal cords take place hand in hand with mass movements and the "up and down" movements of the cords.

The authors point out that the vocal cords during phonation move in both longitudinal and diagonal directions, which movements correspond closely to previous descriptions given by Husson and Tarneaud.[22] [A description of the vertical movements apparently appeared in 1938 in an article by Hartman,[23] the exact title of which I am unable to determine, and in a journal which I have not been able to obtain.—J. P.]

Further observations upon the details of vocal cord movements are brought to light in dramatic and convincing fashion by perhaps the outstanding contribution of the year to the study of laryngeal physiology. This is the motion picture film prepared by the Bell Telephone Co., of New York.[14] Taken by especially built equipment, at varying speeds up to 4,000 frames a second, or 1/250 of normal speed, it demonstrates in most dramatic fashion the movements of the vocal cords as they produce tones at different pitch levels and at varying pressures of expired air. The conclusions drawn from such observations can hardly be questioned and must be recognized as well established principles. In these films the pitch and intensity of the "voicing" has been varied but the vowel used was universally "ae." The pitch varied from 120 to 350 cycles per second, and the intensities from soft to rather loud conversational speech.

At low pitch the film shows the vocal cord movements to be very complex, becoming less so as the pitch is elevated. At very low pitch the vocal folds are completely relaxed. As the pitch is raised, the folds become firmer due to contractions of the underlying thyroarytenoid muscles, and are stretched to greater length. At low pitch, assuming that the cords are in the closed position of the cycle, they begin to open from underneath, the opening progressing upward and outward. After they have opened, the closing phase similarly begins from underneath. As the folds open and close, a wave-like motion or ripple is seen to pass over the top surface from the glottis towards the lateral walls of the larynx. [It is quite impossible to accurately picture these complicated movements

from any written description Only by repeated studies of the film itself can these movements be appreciated.—J. P.]

At low pitch the folds may be nearly closed for half the cycle, and this relative period of closure (during the individual vibration), becomes smaller as the pitch is elevated.

At "extremely high" pitches, only the edges of the cords nearest the glottis are seen to vibrate, so that the variations in the width of the chink of the glottis during vibrations is very slight. The vibration also tends to be confined more and more towards a diminishing portion of the length of the cords as the pitch is increased. [The remainder or nonvibrating segments of each cord being damped by tight approximation with its fellow. This is known as the "falsetto" mechanism. The term is very badly chosen — a far more descriptive term would be the "dampening" mechanism, which I discussed briefly in last year's review of the literature.[17] At any rate, the mechanism in question has been first described and photographically illustrated by Thomas French almost 60 years ago.[18] Its demonstration, therefore, in this film presents nothing new.—J. P.] At high intensities and high pitch, the length of time during which the cords remain tightly closed is smaller than at low pitch. In fact, in "falsetto," complete closure is usually not attained. [Undoubtedly this refers to the anterior vibrating portions of the cords. In all "falsettos" the posterior segments remain firmly approximated and do not vibrate.—J. P.] At these high pitches, however, compression waves may still be seen to traverse the width of the folds. The width of the opening between the folds, the chink of the glottis, becomes narrower than at lower pitches.

With pitch remaining constant and intensity of sound changing, certain variations are noted. At very low intensities the cords close together very feebly or may not close at all. At higher intensities but at the same pitch, they close more firmly and may remain closed for an appreciable time. The widest opening during the cycle does not differ greatly with intensity change. [The authors here are speaking not of abduction and adduction but of the actual vibrations of the cords. The above most important description of changes noted in variations of intensity at fixed pitch are readily explained. Note that the authors point out the widest point of opening does not vary a great deal, but with higher inten-

sities the vibratory closure becomes more complete. In other words, the vibratory movement is over a greater distance (*i.e.*, it more nearly approaches the midline from the same lateral position) when the intensity is increased. Stated more simply, then, *the amplitude of the vibration becomes greater* as the pressure is increased. Curry, in his new text,[19] says "increases in the air pressure, above the minimal value necessary to initiate vibration at a given frequency, determine the amplitude of the vibration, and hence the intensity of the sound produced . . . following roughly the square-law principle." To summarize this, then, the Bell Telephone Co.'s description and demonstration of changes in the vibratory excursion of the cords with varying intensities is simply a more detailed description of the fact that increases in amplitude of vibratory excursion result from increases in pressure, and that this increase in amplitude is represented by a closer approximation to the midline. Despite the weight of evidence, these seemingly simple principles cannot be accepted at their face value. There is more to this problem than meets the eye. It would seem from the evidence produced by the description of the films that when we want to produce louder tones, but without varying the pitch, we simply increase the pressure of expiratory air, the cords move over a wider range and the desired result is produced; however, there is bound to be a further change in laryngeal configuration than has been described. If we take any chamber and permit air from it to escape under pressure through a narrowed orifice, a tone is produced, the pitch of which becomes elevated as the pressure is increased. Very likely the same thing takes place, to a certain extent at least, in the larynx when air from the lungs is forced through it. To maintain a fixed pitch, therefore, with an increase in pressure, some compensation must take place in the larynx to permit maintenance of this fixed pitch in the presence of factors (the increased air pressure) which tend to elevate it. The larynx, therefore, compensates by undergoing a change ordinarily calculated to lower the pitch, but in this instance the pitch does not actually become lower because pressure of air escaping through the larynx from below has been increased. The laryngeal changes which take place to compensate for the increased pressure and which occur in order that the pitch may be maintained while volume is increased may be a diminished tension or thickening of the cords, a foreshortening, or a less pronounced "fal-

setto" mechanism. Such compensatory changes in the larynx are not described by the authors.—J. P.]

Cole[9] writes on laryngeal spasm and so-called tracheal collapse. During a thyroidectomy a patient developed severe stridor despite the fact that the recurrent laryngeal nerves had not been interfered with. It was noted that the trachea was reduced in size to that of a lead pencil and was bloodless. After a few minutes when the stridor disappeared the trachea was unmistakably large and now perhaps the size of the index finger. Experiments were performed to study this phenomenon.

Histologic studies demonstrate that the trachealis muscle is attached to the tips and adjacent portions of the tracheal rings and the fibroelastic membrane between them. Contraction of this muscle appreciably narrows the size of the tracheal lumen, especially if the cartilaginous rings have, for one reason or another, become thinner. Such thinning by erosion is present in certain thyroid enlargements from pressure of the thyroid tumor on the upper tracheal ring.

Stimulation of the trachealis muscle of the dog with electric currents produced a definite narrowing of the tracheal lumen. In the human the trachealis muscle, like most of the laryngeal muscles is supplied by the recurrent laryngeal nerve.

In the larynx, it is pointed out that the adductor muscles of the cords are greater in number and more powerful than the abductors. Stimulation of the recurrent nerve, therefore would result in action controlled by the adductors. In addition, the adductor fibres in the recurrent laryngeal nerve are larger and more readily excited than the abductor fibres. The latter in addition more readily lose their ability to respond to repeated stimuli than do the adductor fibres. For these reasons, stimulation of the recurrent laryngeal nerve directly or by reflex is apt to cause an adductor spasm of the larynx, with consequent stridor rather than an abductor spasm. This reflex can be produced experimentally by such methods as tension on the thyroid gland, crushing or manipulation of the gland, trauma to surrounding blood vessels, lightening of the anesthesia or the sudden administration of anesthetics such as ether. The fact that the stimulation gives a bilateral response is due to a direct crossing of the nerve fibres at the

periphery (within the larynx) and need not be the result of another reflex of central origin. [This is a startling statement. If it were true, I should think severing of one recurrent laryngeal nerve would not result in complete paralysis of muscles supplied by it, since impulses could then pass down the second nerve and cross peripherally to activate the muscles on the side of the severed nerve. We know, however, that this does not occur.—J. P.] This the authors believe is the cause of respiratory collapse during operations upon the thyroid gland. The tracheal collapse is due to the development of negative intracheal pressure within the trachea as a result of the obstructed respiration at the larynx. This, however, occurs to an appreciable degree only when the tracheal rings are thinned from pressure or generally debilitated states. Occasionally, however, the authors point out that this tracheal occlusion may be due directly to the constricting action of the trachealis muscle, because of reflexes passing through the recurrent laryngeal nerve, but this occurs only when the tracheal rings have been thinned or eroded by the pressure of an enlarged thyroid mass. It is interesting to note, as Brewer, quoted by the author, points out, this reflex spasm of the larynx can be initiated by stimuli elsewhere than in the neck. For instance, experimental traction on the mesentery or stimulation of the splanchnic nerve with Faradic current may result in this reflex (adductor) laryngeal spasm. Further proof that this reflex effect actually takes place results from the observation that action potentials in the recurrent laryngeal nerve can be obtained while visceral traction or electric stimulation of the viscera are employed.

Following injuries to the recurrent laryngeal nerve and paralysis of a vocal cord, Jeschek[10] finds that there remains a certain residual movement of the involved arytenoid cartilage. After about six months, however, the fixation of the arytenoid becomes complete.

[This residual movement is possibly due to the action of the interarytenoideus muscle, which is supplied not entirely by the recurrent, but at least partially by the superior laryngeal nerve. The action of the nonparalyzed portions of the interarytenoideus results in mintue movements of the cord until fibrosis of the muscles supplied by the recurrent laryngeal takes place. When fibrosis of the majority of the abductor and adductor muscles has finally occurred, the puli of the

relatively small interarytenoideus is no longer sufficient to move the arytenoid cartilages against the fixation of the fibrosed muscles and the cord at this stage becomes immobile.—J. P.]

A case of congenital double true vocal cord is cited and other literature on the subject quoted by Frank and Malev.[11] Apparently from time to time there are observed reduplications of the true vocal cord, one below the other and separated from each other by a cleft, but joined at each end. These are present in addition to the false cord. Both true cords are covered by stratified squamous epithelium, which, however, is absent in the cleft itself. The fibrous layer in the submucosa dips with the cleft to follow the entire free margin of the cords. This doubling can occur in one or both cords. The condition is a congenital anomaly. Its clinical importance lies in the fact that it leads to a lifelong hoarseness of greater or lesser degree. This is primarily due to faulty laryngeal closure during phonation but may be accompanied by redness and swelling, which likewise play a rôle in the huskiness of the voice.

Guthrie[2] points out that the larynx is probably the only organ of the body treated without employment of functional tests. It is, however, only part of the vocal mechanism. For production of voice, a strong, prolonged, controlled act of expiration is first necessary. This is secured by reinforcing the upward passage of the diaphragm and the downward movement of the ribs by contraction of the abdominal musculature ("abdominal press"). These movements can be studied by a pnemogram obtained by use of a "pnemograph." The laryngeal contribution is next considered. The vocal cords, with the thyroarytenoid musces as their foundation, are adjusted in their shape, bulk and consistency by the aid of all the laryngeal muscles. The actual vibration of the cords is not a muscular effort but an aerodynamic phenomenon, and cannot be studied by larynoscopy or by cinematography. Only by stroboscopy can the vibratory movement be followed as a slow motion picture. [This erroneous concept will be discussed in a subsequent paragraph.—J. P.] The third component of the vocal mechanism is the resonator. These are: *1.* buccal, and *2.* pharyngeal. Each is capable of wide variations in size and shape. Without these the laryngeal note is weak and indefinite.

The cry of an infant corresponds to 435 cycles and the range gradually extends, but is very limited until puberty. Then the cords lengthen and the male voice undergoes the alteration known as the "break." The pitch is then lowered to that of the adult voice. In girls, the vocal change of puberty is much less marked. A redness and slight swelling of the vocal process is common at this time and may lead to an erroneous diagnosis of laryngitis. In incomplete mutations a high-pitched voice may persist and is easily treated by gentle backward pressure on the thyroid cartilage during the production of a low note and by suitable humming exercises. Cases due to arrest of sex development are less favorable and must be treated with hormones.

An individual adult voice extends over two and one-half octaves, and the pitch used in speaking lies at the lower part of the singing range. Incorrect classification of compass (for example, a baritone who believes he is a tenor) is one of the commonest causes of vocal failure. [This is one of the reasons why I object to such an artificial classification of voices as is commonly employed. A vocalist should sing only within his easy range, and not attempt to "reach," just because he classifies as a tenor, and such higher or lower tones are expected of a tenor.—J. P.]

The method by which a note is initiated is of the greatest importance in vocal hygiene. All teachers agree on advising the crescendo method of "swelling on a note" and in condemning "glottal shock."

The various "registers" have provoked much discussion. The "chest," "middle" and "head" registers are named according to the subjective sensation of the singer and do not imply that the voice is produced at such levels. There is a change of vocal quality as the singer passes from one register to another. The position of the larynx during this passage may alter, and there are usually changes in the form and tension of the vocal cords.

In vocal dysfunction the complaints may be weakness of the voice, a tendency to "crack" at certain pitches, recurrent hoarseness, dryness, burning, tightness and pressure, increased by use of the voice. When examining the throats of these people it is important to realize that a dry mucosa, lymphoid nodules or dilated vessels may be the result and not

the cause of the vocal trouble. The singer should be asked to sing, and the vocal range, the mode of attack and the method of breathing noted. Orthophonic or re-educative treatment (chiefly the duty of the singing teacher, in which the laryngologist co-operates) may be by use of the "Harmonic Vibrator," an instrument which transmits to an applicator held against the front of the larynx, audible vibrations resulting in specific tones which may be felt as well as heard. With the apparatus in action, it is easy to sing a corresponding tone; indeed, it is unpleasant and even painful to sing out of tune with the vibrator. Local treatment is secondary to the above methods of treating errors of function. The psychological factors involved, such as fear and anxiety starts and other neuroses, must not be overlooked, and if these are present should be treated by suggestion. General hygienic measures to assure sound body are important. Local treatment to the larynx is not important. Local physiotherapy to the larynx, such as cold compresses and massage, may be of value.

Prof. R. W. Johnstone, in discussing this paper, pointed out that a female singer inevitably retired when pregnant because of a certain change in the voice, generally supposed to be due to a thickening of the mucous membrane. Guthrie agrees that the voice altered during pregnancy.

In a discussion of functional disorders of the voice, Guthrie[12] repeats that the larynx is only part of the vocal mechanism. The organs concerned in breathing, articulation and resonance must also be considered. The vocal cords with the thyroarytenoid muscle as their foundation are adjusted by the aid of the laryngeal muscles in their shape, bulk and consistency. The actual vibration of the cords is not a muscular effort but an aerodynamic phenomenon and cannot be studied by laryngoscopy or by cinematography. Stroboscopy, Guthrie says, is the only method by which these vibratory movements can be followed. [This latter statement is, of course, written without Dr. Guthrie having seen the Bell Telephone Co.'s slow-motion laryngeal pictures or my own taken in slow motion. Both of these films adequately demonstrate such vibrations. Furthermore, cinematography is probably a more accurate method of observing vocal cord vibrations than is observation through a stroboscope. The motion pictures demstrate every phase of a single movement or of repeated movements, whereas a stroboscope interrupting light at, let us say,

127 vibrations a second and permitting only 1/128 part of each vibration to be visualized, requires 128 different vibrations to show a single complete cycle. The resultant stroboscope picture would probably represent a true reproduction of cord movements if it were certain that the vibratory movements of the vocal cords were taking place exactly at the anticipated speed and that each of 128 vibrations were produced with exactly the same pressure and muscular tension. If, however, these movements were taking place a little more or less rapidly than 128, as could easily occur in the ordinary routine examination, a certain distortion would undoubtedly take place. It seems to me that unless this speed of vibration and numerous other factors as well were controlled by the most careful physical analysis, a stroboscope picture may vary somewhat from the true state of affairs.—J. P.]

Guthrie repeats his observations previously reported that there are two chief resonating chambers, buccal and pharyngeal, which act as a mobile loud speaker horn. Without this resonating effect the laryngeal tone is weak and indefinite, as shown by experimental observation and in cases of cut throat.

Sir Richard Paget in his part of the discussion dealt with "phonation of the vocal cords." These behave exactly like the lips of a trumpeter when blowing a trumpet. He describes the large number of tones and variations in quality which can be produced when blowing a trumpet entirely by adjustment of the lips. The vocal cords, he says, do exactly the same thing. [In slow motion pictures of the larynx taken by Mr. Hinman and myself, it is astonishing to note the similarity in appearance between vocal cord movements in slow motion and a trumpeter's lip movements at normal speed. If the projector be placed on its side so that the projected vocal cords lie horizontally across the screen instead of vertically, and, therefore, in the position in which we are accustomed to observe lip-movements, the movements of the trumpeter's lips and the vocal cords appear to be very much alike.—J. P.]

Sir Richard Paget continues and points out that if a slit in the wall of a rubber tube be made and air blown through the slit, the latter would not vibrate like a reed unless the vibrating part was coupled with a resonator which helped it. Without the resonator the slit remained open continuously without vibrating. The equivalent of the resonator and,

therefore, an important factor in the production of tones from the human vocal cords, Paget believes to be the false vocal cords which represent a resonating cavity, the volume and aperture of which give the necessary variety of "resonant pitch." [Objections might certainly be raised to this point of view. The most obvious of these objections is perhaps Guthrie's observations just quoted of the lack of resonance in voices of persons with cut-throats, in which a fistula was created below the pharynx, but above the larynx. The false cords were below the fistula and free, therefore, to exert whatever qualities of resonance they might produce. The voice, however, in these instances is characteristically without volume or resonance and produces a weak, indefinite tone, which would tend, therefore, to rule out any great rôle the false cords may play as resonators. Besides, there seems to be no great difference (as Negus points out in his text, "The Mechanism of the Larynx") in the resonance or quality of tones produced by cats and human infants. Yet the cat has no false cords at all, the interior of the larynx consisting solely of a single thyroarytenoid fold. The resonant qualities of the voice of the cat in the absence of any false vocal cords would most certainly mitigate against the belief that false vocal cords acted in any important way as resonators. They very likely, in addition to other functions, play a more important rôle in the production of overtones.—J. P.]

Paget points out, in returning to the analogy of the trumpeter, that if the pitch of the trumpet is high, the trumpeter uses a small mouthpiece, and in trumpets of low pitch the mouthpiece is larger. [Very likely Paget intends this to demonstrate an analogy between the size of the trumpet mouthpiece and the chink of the glottis. In both instances the smaller the lumen through which air passes under pressure the higher is the pitch produced. This in the larynx is well demonstrated in the contour adopted during damping of the cords (erroneously called "falsetto-voice"), in which a tiny orifice produces an extremely high tone, and the larger orifice with less damping produce lower tones.[13]—J. P.] Paget further suggests an added function of the false vocal cords, which he says is "quite as certain as their function in producing resonance." [This seems a little ambiguous, especially since false vocal cord function as it applies to resonance remains considerably in doubt.—J. P.] This function relates

to the difference in the production of, for example, P and B, and S and Z. Apparently Paget made a model in which with the same mouth closure that produced P could be utilized to produce B. If both the true and the false cords were equally closed or open, B was produced, but if either the true or the false cords in the model were widely opened and the other shut, P was produced. The author evidently concluded that this placed upon the action of the false cords the possible function of determining whether an articulated sound was to be a P or B, or S or Z, or F or D. The difference (in the model) was one of resonance. [For general acceptance, I should think that a concept so revolutionary would require more proof than its application in a model, which may or may not create a state of affairs similar to that in the human. We are generally accustomed to think of the larynx as a producer of sound of varying pitch and intensity, but not particularly related to the conversion of that sound into vowels or consonants, a function which is ordinarily ascribed to the action of the mouth, tongue, etc.; however, in our present state or knowledge we are in no position to deny the observations and conclusions of Paget, although the burden of proof most certainly rests upon him.—J. P.] Paget tells of having examined the larynx of G. O. R. with the laryngeal periscope and observing that when the subject smiled or scowled "along with this change in facial expression there was an obvious change in laryngeal expression." He believes that "Nature in this way provided for the expression of emotion by night and by day — the visual expression was quite adequate during the daytime, and the laryngeal expression gave corresponding characteristic sounds by which the emotions could be recognized during the hours of darkness!"[J. P.]

Robert Curry, entering the above discussion, commented upon Guthrie and Milner raising the issue of the "attack" of a given tone in singing, and points out that this is connected with the synchronization of the beginning of expiration and the closure of the vocal cords for phonation. Inspiration involved opening of the glottis, but when voice sounds can be produced during the inspiratory phase of respiration this was called a "low-voice press." It is accompanied in the lowest pitches by depression of the larynx in the neck and a pushing back or drawing-in of the chin and efforts to retract the jaw. The production of highest tones is accompanied by the reverse action.

Curry in this same discussion objects to the simile between the speech apparatus of the human being and the violin string. This, he says, gives rise to the mistaken impression that the vocal cords are stretched during the production of higher and higher pitches. No such stretching action, he says, takes place, or at the most a few millimetres. [Actual measurements of the stretching of the vocal cords are not available, but from repeated observations of the Bell Telephone Co.'s films it would appear that such stretching is very appreciable. This is indicated in a publication[14] issued by the company itself and substantiated by my own personal observation of the film upon which they base their deductions.—J. P.] Curry points out that the vocal cords vibrate not individually but as a part of a coupled system, including the false cords, the ventricles and the pharynx and oropharynx. In any uttered sound, this speech system acts as an entity and its entire contour changed for every variation in vocalization.

Cyril Hosford closed this discussion before the Royal Society of Medicine by pleading for closer collaboration with the singing master, which would cure abnormalities of the voice by the removal of the faults which caused them.

Freedman[15] presents an interesting resumé of some of the functions of the laryngeal ventricle and its appended saccule. This latter, as John Hilton[16] pointed out in 1837, is an anterior-upward projection of the horizontal slit of the ventricle of Morgagni and forms an anterior or blind pouch, known as the sacculis ventriculi laryngis. Freedman adds to this the description of Irwin Moore,[24] who points out that "the sacculus lies between the ventricular band and the inner surface of the thyroid cartilage. It extends directly upwards from the anterior part of the ventricle and represents the air-sacs which in anthropoid apes are connected with the ventricles. The sacculus varies greaty in size and shape; sometimes it is nearly conical with its base below, in other cases it is pear-shaped with the broader part above. It averages about half an inch in the vertical direction, reaching the upper border of the thyroid cartilage. The thyroepiglottideus muscles and aryepiglottideus muscles [fibres of the thyroarytenoideus and the interarytenoideii, respectively, which are sometimes, especially in older works, considered as independent muscles.—J. P.] as they sweep into the aryepiglottidean fold sur-

round the upper part of the sacculus and act as the Compressor Sacculi Laryngis of Hilton."

I have quoted this anatomical description of the ventricle saccule in detail because it is necessary to bear this in mind when considering Freedman's description of its function. He points out that the sacculus is invested throughout with mucous glands. These glands secrete a thin mucus which remains in the sacculus until the latter is compressed by the action of the "Compressor Sacculi Laryngis" described above. This thin secretion is then poured over the vocal cords, acting as a lubricant. Two crescentic folds at the mouth of the sacculus where it enters the ventricle are also described by Hilton, who ascribes to them the function of breaking the stream of fluid from the sac and giving it a general diffusion over the surface of the vocal cords. [Or more likely acting as a valve to retain appreciable amounts of secretion in the reservoir of the saccule until such time as it shall be needed, whereupon the pressure of the Compressor Sacculi will force it out against the resistance of the valve. —J. P.]

The sacculus, Freedman continues, may thus be considered the "oil can of the vocal cords." During phonation or deglutition the sphincters (adductors) of the larynx are put into action. Thus, when the vocal cords vibrate against each other and need lubricant most, the compressor sacculi is set in motion and squirts out the lubricant. [In Mr. Hinman's and my own motion pictures of the larynx, one sequence demonstrates the truth of these observations. Even though the patient is recumbent, and the anterior portion of the larynx is uppermost, nevertheless secretions which accumulate upon the vocal cords first appear near the anterior commissure at the point where the sacculus empties. It can be seen that the lubricating fluid accumulates periodically at this point in relatively large amounts and then pours over the remainder of the cords. It definitely, as the article describes, gives the impression of being projected onto the vocal cords "in squirts."—J. P.]

Freedman discusses the phylogenesis of the saccule in man. He believes it to be an evolutionary degeneration of much larger air sacs which lead out of the ventricle with a function directed to rebreathing air. Such huge air sacs are still found

in the orang-utan and the chimpanzee, extending down the neck through the thyroid membrane between the sternohyoid muscles, spreading out at the root of the neck, sometimes reaching actually into the axillae. The function of these enormous air-sacs in anthropoids is bound up with bringing into action of the sphincter mechanism of the larynx, and the consequent necessary cessation of respiration during powerful use of the forelimbs, which usage requires a fixed thoracic cage. During this forced cessation of respiration in the presence of great physical activity, the air sacs become a convenient source of oxygen and act as respiratory mixing chambers.

Taft[20] describes the simplicity of obtaining equipment capable of providing fine oscillographic tracings of the voice. The cathode ray oscillograph is in use in practically every well equipped radio repair shop. It is only necessary to have a microphone to pick up the voice, a public address system to build up the power to the necessary intensity, and the ordinary radio-shop oscillograph on the screen of which the patterns appear. These patterns are then photographed with an ordinary camera capable of close-up focusing, and satisfactory oscillographic tracings thus obtained.

[Even though all the published articles of the latter months of 1938 and 1939 could not be obtained in time for this resumé, the complete bibliography is nevertheless appended. Articles not reviewed appear at the end of the list and have no reference number preceding them. They will be reviewed in 1941.—J. P.]

BIBLIOGRAPHY.

1. Appelbaum, I. L.: The Respiratory Defense Mechanism. *Amer. Jour. Med. Sci.*, 198:356-366, Sept., 1939.

2. Guthrie, D.: Notes on Physiology and Pathology of Voice. *Trans. Med.-Chir. Soc., Edinburgh Med. Jour.*, March, 1939.

3. King, B. T.: Bilateral Abductor Cord Paralysis. *N.W. Med.*, 38:125, April, 1939.

4. Jackson, C., and Jackson, C. L.: Your Voice. *Hygeia*, 17:110-112, Feb., 1939.

5. Jannulis, G. E.: Experimental and Therapeutic Results Dealing with the External Branch of Superior Laryngeal Nerve in Relation to the Function of Cricothyroideus Muscle. *Monatsschr. f. Ohrenheilk.*, 73:64-65, Jan., 1939.

6. Trendelenburg, W.: Expression of Labium Vocale Movements in Vocal Curve. *Zischr. f. Hals., Nasen. u. Ohrenheilk.*, 44:76-78, 1938.

7. ZERFFI, W.: Functional Vocal Disabilities. THE LARYNGOSCOPE, 49:1143-1147, Feb., 1939.

8. HARTMAN, W., and WULLSTEIN, H.: Examination of Motor Processes of Vibrating Labia Vocalia of Laryngeal Preparations Examined by Improved Photoelectric Cell. *Arch. f. Ohrenheilk., Nasen. u. Kehlkoph.*, 144:348-360, 1938.

9. COLE, W. H.: Laryngospasm and Tracheal Collapse During Thyroidectomies. *Arch. Surg.*, 39:10-27, July, 1939.

10. JESCHEK, J.: Movement of Arytenoid Cartilage in Paralyzed Vocal Cord: The Problem of Recurrent Paralysis. *Arch. f. Ohrenheilk., Nasen. u. Kehlkoph.*, 145:315-321, 1938.

11. FRANK, DAVID I., and MALEV, MILTON: Double Vocal Cord. *Arch. Otol.*, 29:713-716, April, 1939.

12. GUTHRIE, DOUGLAS, and MILNER, AUGUSTUS: Discussion on Functional Disorders of the Voice. *Jour. Laryngol. and Otol.*, 54:261-271, May, 1939.

13. PRESSMAN, J. J.: Action of the Larynx. *Reprint from Proc. Roy. Soc. of Med., Jour. Laryngol. and Otol.*, 53:672, Oct., 1938.

14. Bell Telephone Laboratories, New York. High Speed Motion Pictures of the Human Vocal Cords.

15. FREEDMAN, A. O.: Diseases of the Ventricle of Morgagni. *Arch. Otolaryngol.*, 28:328-343, 1938.

16. HILTON, J.: Description of the Sacculus or Pouch in the Human Larynx. *Guy's Hosp. Rep.*, 2:519, 1837.

17. PRESSMAN, J. J.: Physiology of the Larynx. THE LARYNGOSCOPE, 49:239-259, April, 1939.

18. FRENCH, T.: *Proc. Amer. Laryngol. Soc.*, 1886.

19. CURRY, ROBERT: Mechanism of the Human Voice, p. 47. Longmans, Green & Co.

20. TAFT, ROBERT B.: Use of Oscillograms in Studies of the Voice. *Arch. Otalaryngol.*, 28:999-1000, 1938.

21. NEGUS, V. E.: The Mechanism of the Larynx. Wm. Heineman Co., London, 1929.

22. HUSSON and TARNEAUD: *Rev. de Laryngol.*, 53:II:64, 1932.

23. HARTMAN, W.: *Arch. f. Sprach. u. Stimmheilk.*, 2, 1938.

24. MOORE, I.: The So-Called Prolapse of the Laryngeal Ventricle and Eversion of the Sacculus. *Jour. Laryngol. and Otol.*, 37:265, 333 and 381, 1922.

O'GORMAN, F. J. P.: Laryngeal Vertigo. *Brit. Med. Jour.*, 1:271, Feb. 11, 1939.

BEAU, A.: Process of Ossification of Cartilages of the Larynx. *Rev. Med. de Nancy*, 67:127-136, Feb. 15, 1939.

EVANS, T. H.: Anatomy and Histology of the Larynx — Function of Infantile Rate of Respiration. *Bull. N. Y. Med. Col.*, 2:29-32, April, 1939.

NEMAI, J.: Larynx of a Professional Singer. *Gyogyaszak*, 79:4, Jan., 1939.

CALVET, J.: Change of Voice and Early Macrogenitosomia (Case). *Rev. franc. de phon.*, 7:101-103, April, 1939.

MOTTA, R.: Changes in Voice After Castration (Experiment on Cocks). *Valsalva*, 14:478-483, Oct., 1938.

DESSY, G.: Respiratory Movements of the Larynx. Description of Apparatus Used for Registration. *Riv. di pat e clin. d. tuberc.*, 12:791-806, Nov. 30, 1938.

KREINDLER, A.: Rhythmic and Synchronous Myoclonias of Pharyngeal Cavity and of the Larynx. *Schweiz. Arch. f. Neurol. u. Psychiat.*, 43:79-88, 1939.

REDOGLIA, F.: Case of Laryngeal Paralysis Resulting from Intracranial Lesion of the Vagal and Spinal Accessary Nerve. *Valsalva*, 14:566-573, Dec., 1938.

GUTHRIE, D.: Physiology of Vocal Mechanism. *Brit. Med. Jour.*, 2:1189-1195, Dec. 10, 1938.

MITRINOWIC, A.: Phonologic and Stroboscopic Examination of the Larynx in Phonasthenia. *Medycyna*, 793-796, Nov. 7, 1938.

STRUPPLER, V.: Innervation of the Larynx. *Ztschr. f. Anat. u. Entwecklngsgesch*, 109:360-373, 1939.

PAULY, R.: Associated Paralysis of Dilator Muscles of the Glottis: Anatomicoclinical Study. *Rev. de laryngol.*, 60:349-353 April, 1939.

CANUYE, G., and GUNSETT, A.: Planigraphy of Face of the Larynx. *Jour. de radiol. et d'electrol.*, 23:193-202, May, 1939.

MOLLISON, W. M.: Loss of Voice. *Guy's Hosp. Gaz.*, 53:196, June 17, 1939; 213, July, 1939.

WICART, A.: Relation Between Speaking and Singing Voice. *Valsalva*, 14:317-331, July, 1939.

RONCALLO, P.: Anatomy and Histology of the Larynx. Insertion of the Vocal Muscular Fibres in the Vocal Ligaments. *Arch. ital. di anat. et di embriol.*, 41:357-367, 1939.

MACMAHON, C.: Treatment of Dysphonia and Allied Conditions. *Jour. Laryngol. and Otol.*, 54:343-359, June, 1939.

HALA, B.: Oscillographic Analysis of Laryngeal Voice in Relation to the Formation of Vowels. *Valsalva*, 14:332-345, July, 1938.

WUSTHOFF, P. G.: Significance of Heredity in Quality of the Voice. *Jahresk. f. arztl. Footbild.*, 29:38-51, Nov., 1938.

HEATLY, C. A.: Larynx in Infancy. *Arch. Otolaryngol.*, 29:90-103, Jan., 1939.

HEATLY, C. A.: Chronic Stridor in Infancy. *Arch. Otolaryngol.*, 29:90-103, Jan., 1939.

TAFT, R. B.: Use of Oscillograms in Laryngeal Studies. *Arch. Otolaryngol.*, 28:999-1000, Dec., 1938.

FREEDMAN, A. O.: Anatomy and Histology of the Larynx. *Arch. Otolaryngol.*, 28:329-343, Sept., 1938.

1917 Wilshire Boulevard.

TYPE III PNEUMOCOCCUS MENINGITIS OF OTITIC ORIGIN. RECOVERY FOLLOWING TREATMENT WITH SULFAPYRIDINE.*†‡

DR. MATTHEW S. ERSNER, DR. DAVID MYERS and
DR. MERRILL B. HAYES, Philadelphia.

During the past year, several cases of recovery from pneumococcic meningitis following the use of sulfapyridine 2-(p-aminobenzenesulfonamido) have been reported by Reid and Dyke,[1] Cunningham,[2] Robertson,[3] Cutts, Gregory and West.[4] Hodes, quoted by Perrin H. Long,[5] reported four cases of pneumococic meningitis treated at the Sydenham Hospital in Baltimore. One case recovered, and of the three that died, life in two seemed to have been definitely prolonged.

In view of the rarity of recovery from pneumococcic meningitis, despite the multitude of treatments that were recommended in the past, the following case is reported.

S. S., male, age 15 years, was admitted to the Temple University Hospital on Dec. 19, 1938, with a history of a right suppurative otitis media of five weeks' duration. Two weeks prior to admission, there was a swelling over the right zygomatic region. An immediate mastoidectomy was done, which revealed extensive necrosis and contraction of the mastoid tip and a marked amount of destruction of the zygoma. The lateral sinus and dura of the middle fossa were exposed, due to the erosion of the plate of the middle fossa dura and the lateral sinus. A culture made at the time of operation was reported as pneumococcus type III.

The patient did well postoperatively (see Fig. 1) until the ninth day, when he developed a high temperature, and a meningeal complication was diagnosed. The spinal fluid revealed an increased cell count, and on smear and culture, pneumococcus type III was obtained. The temporal bone was

*From the Department of Otology, Temple University Hospital, Service of Dr. Matthew S. Ersner.

†The sulfapyridine used was supplied by Merck and Co., Rahway, N. J.

‡Read before the Philadelphia Laryngological Society, March 7, 1939.

Editor's Note: This ms. received in Laryngoscope Office and accepted for publication, June 15, 1939.

X-rayed and indicated an involvement of the right petrous apex (see Figs. 2, 3 and 4).

On this day, Dec. 28, 1938, nine days after the first operation, a mastoid revision and a Lempert[6] apicectomy were performed. No pus was found, but there was definite necrosis of the cells at the apical carotid portion of the right temporal bone.

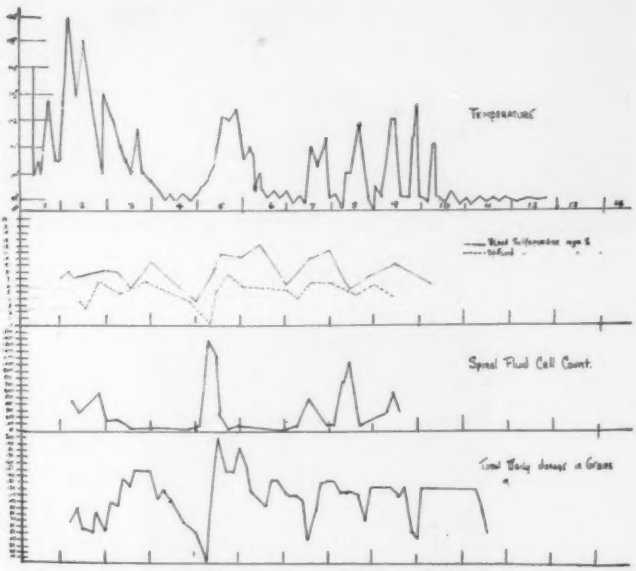


Fig. 1. Graphic representation of 12 weeks of illness. Block 1: Temperature from time of admission to hospital to end of twelfth week of hospitalization. Block 2: Concentration of sulfapyridine in blood and spinal fluid (Marshall). Block 3: Spinal fluid cell count. Block 4: Total daily dosage in grains of sulfapyridine.

Note: In fourth week, the temperature was normal, sulfapyridine was reduced, the spinal count was normal and the concentration of blood and spinal fluid dropped. In the fifth week, the recurrence of symptoms. Note the rise in temperature and rise in spinal fluid count. The dose of sulfapyridine was immediately increased to a high level, with a resultant rise in the blood and spinal fluid concentration and almost an immediate drop in the temperature and spinal fluid cell count.

The patient was immediately given sulfapyridine orally, and in the next few days a remarkable improvement ensued. The spinal fluid became clear and the temperature normal. When the boy seemed practically recovered, the sulfapyridine dosage was reduced and again a rise in temperature occurred,

with a reappearance of meningeal symptoms and cells and organisms in the spinal fluid. The dosage of sulfapyridine was immediately increased and again clinical improvement

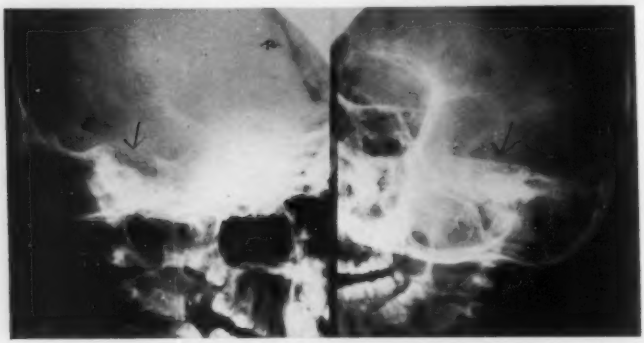


Fig. 2. X-ray of petrous portion of temporal bone. Right and left for comparison. Right side shows the area of bone removed at the mastoidectomy and, in addition (at arrow), the involvement of the petrous apex.

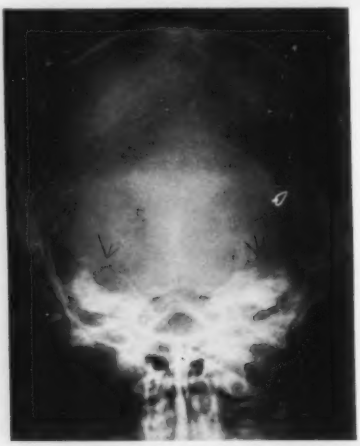


Fig. 3. Coronal view. Note erosion and absence of structure at the apical carotid portion of the right temporal bone as compared to the left.

occurred. When the sulfapyridine was decreased, there was a recrudescence of symptoms. The patient had been receiving as much as 150 gr. of sulfapyridine daily. The accompanying

chart reveals the relationship of the temperature, spinal fluid findings and dosage of sulfapyridine. The total intake of the drug over a period of 10 weeks was more than 6,952 gr., approximately 463.49 gm.

The frequent return of meningeal symptoms following the reduction in sulfapyridine dosage indicated that drug therapy alone was not sufficient to eradicate the meningeal infection. At the suggestion of Dr. F. W. McNair Scott and Dr. N. Kendall, of the Pediatric Department of Temple University, a Francis test was done with negative result, so that on Feb. 4, 1939 (38 days after the onset of the meningeal symptoms), 20,000 units of type III antipneumococcic rabbit serum was

Fig. 4. Note the appearance of the petrous portion. The perilabyrinthine cells completely exenterated so that the semicircular canals are plainly seen. The tegmen of the middle fossa completely removed, and the area of the apical carotid portion that was curetted and drained.

given intravenously. In addition, the patient was given 8 cc. of his own blood serum intrathecally, to supply complement to the spinal fluid. The Francis test was positive after the serum administration. The sulfapyridine was continued as before.

The subsequent course of therapy was as follows: The rabbit serum was administered on four more occasions. The blood serum was given eight more times. The patient received eight whole-blood transfusions. Spinal puncture was done every day until the spinal fluid was clear and free of cells and organ-

isms. Finally, in the tenth week of the illness, the symptoms subsided completely, the temperature became normal and the convalescence became uninterrupted. The sulfapyridine was continued in diminishing dosage during the entire period of hospitalization.

Comment: This case illustrates that, despite the apparent efficacy of the sulfapyridine against the pneumococcus, at times the drug alone may be insufficient to overcome the infection. The addition of the specific sera to the treatment enhances its therapeutic value.

The chart illustrates that in this particular patient the concentration of the sulfapyridine in the blood closely followed the dosage. The boy received large dosage of sulfapyridine over a long period without ill effect. The dose of the sulfapyridine should be regulated by the condition of the patient and the concentration in the blood rather than by a table of dosage.

The complete eradication of the fixed focus of infection in the mastoid and in the apical carotid portion of the temporal bone is always an important part of the treatment of meningeal complications of otitic origin.

BIBLIOGRAPHY.

1. Reid, G. C. K., and Dyke, S. C.: Pneumococcal Meningitis Treated with M and B 693. *Lancet*, 235:619-620, Sept. 10, 1938.

2. Cunningham, A. A.: Pneumococcal Meningitis Treated with Sulfanilamide and M. and B 693. *Lancet*, 235:1114-1115, Nov. 12, 1938.

3. Robertson, K.: Case of Pneumococcal Meningitis Treated with M and B 693. *Lancet*, 235:728, Sept. 24, 1938.

4. Cutts, M.; Gregory, K. K., and West, E. J.: Pneumococcic Meningitis Successfully Treated with Sulfapyridine. *Jour. A. M. A.*, 112:1456-1457, April 15, 1939.

5. Long, P. H.; Bliss, E. A., and Feinstone, W. H.: The Effects of Sulfapyridine, Sulfanilamide and Related Compounds in Bacterial Infections. *Pa. State Med. Jour.*, Feb. 11, 1939.

6. Lempert, Julius: Complete Apicectomy (Mastoidotympanoapicectomy). *Arch. Otolaryngol.*, 25:144-177, Feb., 1937.

1915 Spruce Street.

OTITIC HYDROCEPHALUS.*

DR. HARRY ROSENWASSER, New York.

This case is presented because it illustrates some of the difficulties which obtain in establishing a diagnosis of so-called otitic hydrocephalus.

History: This patient, now age 26 years, was admitted to the Mt. Sinai Hospital 23 years ago with bilateral middle ear suppuration and left mastoiditis. She was operated upon by Dr. Fred Whiting, and an infected cholesteatoma was found. Six years later, the patient had a recurrent left mastoiditis which necessitated drainage. Two months later, a radical mastoidectomy for an infected cholesteatoma was performed by Dr. Maybaum, at which time a large cholesteatomatous mass filling the left middle ear and mastoid cavity was cleaned out. The patient was apparently well except for slight bilateral middle ear discharge until July 24, 1937, when she was admitted for the fourth time. Eight days previously, she had severe right earache with radiation of the pain to the right temporal region, which kept her awake all night. The following day, six days before admission, the patient had a shaking chill lasting one hour, with a rise in temperature to 106°. She noted increased deafness and blurred vision following the chill. For the next five days before admission, there were shaking chills, associated with nausea and vomiting.

Examination: The patient appeared quite sick, was co-operative, the temperature was 105.4°, the fundi oculi were normal, there were no ocular palsies, no abnormal motor or sensory changes, no Kernig sign and no Brudzinski sign. Right ear: There was a thin discharge from a large perforation. The hearing was markedly impaired, there were no landmarks present and there was definite tenderness over the mastoid. Left ear: There was scant discharge from the epithelialized tympanic cavity; the postauricular scar was depressed. There was a fluctuant swelling in the upper end of the scar

*Read at the meeting of the New York Academy of Medicine, Section on Otolaryngology, May 3, 1939.

Editor's Note: This ms. received in Laryngoscope Office and accepted for publication, June 15, 1939.

the size of a cherry. The hearing was markedly impaired. There was no spontaneous nystagmus.

Laboratory Data: On admission, the hemoglobin was 68 per cent; the white blood count was 14,800, with 54 per cent segmented polymorphonuclear leukocytes, 23 per cent non-segmented polymorphonuclear leukocytes, 13 per cent lymphocytes, 4 per cent monocytes, 1 per cent plasma cells and 5 per cent unclassified cells. Daily hemoglobin determinations revealed a steady drop in the hemoglobin. Blood counts repeatedly demonstrated the picture of a severe infection. Blood Wassermann was negative. Culture of the pus from both ears were reported bacillus proteus. Blood sugar, urea nitrogen were normal. Widal, paratyphoid A and B, melitensis and Brucella abortus sepsis were all ruled out by specific agglutination tests. Three successive blood cultures contained no growth. Despite the negative blood cultures, it was the consensus that the patient had an otitic sepsis and that operation was indicated.

Operation: A right complete simple mastoidectomy was performed. There was no evidence of an acute destructive process in the mastoid. There was considerable sclerosis involving the periantral region. Evidence of foul-smelling infected cholesteatoma involving the antrum and middle ear was found. Cheesy, dirty debris was noted issuing from the antrum when pressure was made over the zygoma.

The sinus plate was removed, discoloration of the sigmoid sinus at the knee was found. The sinus was exposed and then packed because of excessive bleeding, without incision or adequate inspection of the lumen of the vein. The internal jugular vein was then quickly ligated. During the operation, the patient was given a transfusion of 500 cc. of citrated blood.

Course: The spiking temperature subsided and remained below 99° for two days. The general condition of the patient seemed good. Nystagmus to the left with nausea and vomiting appeared. Another transfusion of 500 cc. of citrated blood was given. The fundi, which had been normal, became definitely blurred; the following day, the nystagmus, which had been marked to the left, changed and now was directed towards the right. There was no pastpointing, no adiadokokinesis and no ataxia. On the fifth postoperative day, the

packings and plugs were removed from the sinus, which did not bleed, relieving the pressure on the cerebellar dura.

Despite removal of the packings, the nystagmus to the left, the nausea and vomiting persisted, and the patient complained of headache. Bilateral papilloedema (1-1.5 diopters) was now evident. The neurologist stated that "the patient presented evidence of increased intracranial pressure, of meningeal irritation and of signs that were either labyrinthine or cerebellar." Spinal tap at this time revealed an initial pressure below 150 mm., containing eight cells, all lymphocytes with negative Pandy test and no bacteria. The ophthalmologist believed "that the rapid progress of the choking of the discs suggested a posterior fossa lesion." According to the otologist, "the rapid increase in papilloedema, if due to a posterior fossa lesion, should be accompanied by more signs of such a lesion. Not infrequently, papilloedema occurs after only a sinus operation with internal jugular vein ligation. This, however, in sinus thrombosis is not accompanied by such severe headache and vomiting. We may be dealing with two coincident mechanisms, *i.e.: 1.* developing cerebellar abscess, and *2.* the ocular changes following sinus thrombosis." The neurosurgeon stated at this time, Aug. 11, 1937, that "the patient should be watched for further signs of a brain abscess. The few signs at present point to the right temporal lobe. Because of the indefinite localizing signs, the increasing headache, nausea and advancing papilloedema, it was felt advisable to perform ventriculography."

On Aug. 22, 1937, the patient was transferred to the neurosurgical service, and ventriculography was performed by Dr. A. Kaplan. A single trephine was performed only on the left because of the possible danger of infection in performing bilateral trephine, because of infection in the right mastoid wound. The dura was exposed in the usual manner, appeared normal in color, and pulsated rather freely. When the dura was opened, the brain could be seen retracted. The ventricle needle encountered clear fluid, and 40 cc. of fluid was removed and an equal amount of air replaced. X-rays showed little air in the left ventricle, which was not particularly dilated or displaced.

Despite the increased risk, it was necessary to obtain more satisfactory air studies, and therefore a bilateral trephine

ventriculogram was performed by Dr. Kaplan. These Roentgenograms showed no striking abnormality. Repeated fundus examinations indicated subsidence of the papilloedema, repeated lumbar punctures were followed by gradual subsidence of symptoms and it seemed fair to assume that we were dealing with a so-called "otitic hydrocephalus" rather than an otitic brain abscess predicated upon interference with the intracranial circulation. Twelve weeks after admission, the patient was discharged, symptomatically improved but with persistent low grade papilloedema.

Feb. 23, 1938, eight months later, the patient was again readmitted, complaining of bouts of dizziness with changes in position of the head, and the elicitation of severe dizziness when the left ear is pressed against her head. The patient was seen by a neurosurgeon, who could find no evidence in favor of a brain abscess, either temporal or cerebellar. Lumbar puncture was performed. The initial pressure was 280 mg.; 30 cc. of fluid was removed. It was clear and colorless, negative Pandy, and there were only three cells. The papilloedema (3 diopters) was still present. There was a slight nystagmus on looking to the left, which was intensified somewhat by pressure over the upper part of the external auditory canal entrance. Whether this was a true fistula symptom or due to dural pressure could not be definitely decided. All the symptoms again subsided and the patient was again discharged for further follow-up.

On March 28, 1938, the patient was readmitted, again complaining of difficulty in walking, with a tendency to veer to one side or the other, and dizziness. Examination revealed some gait disturbances, some dysdiadokokinesis in the right hand, chronic papilloedema in both eyes, with beginning secondary atrophy. Visual fields showed bilateral enlarged blind spots, and lumbar puncture showed initial pressure of 210 and a final pressure of 80 mm. after removal of only 10 cc. There were only two cells present per c.s. The picture was that of a diffuse labyrinthitis on the left (either a degenerative process or a diffuse latent labyrinthitis), and only by the subsequent course could one differentiate the type of lesion in the labyrinth.

On Jan. 13, 1939, this patient was again readmitted, complaining this time of headache, vertigo and nausea, with pain

in the left frontoethmoid region daily. The ophthalmologist, on Jan. 16, 1939, found o.d. nerve head somewhat hyperemic, the margin indistinct, with slight peripapillary grayness. Arteries were somewhat irregular in calibre. In the macula there was a small area of pigment disorganization. O.s. nerve margins were sharper than o.d., with less peripapillary grayness. There was a small spot of pigment disorganization in the macula. *Opinion:* The nerve heads are not atrophic but there are still some residual changes present.

Spinal tap was again performed and the initial pressure was 260 mm. There was no evidence of block, negative Pandy, and only two cells (monocytes). The patient was seen in the neurosurgical follow-up clinic on March 7, 1939, where she was seen by Dr. Kaplan, still complaining of impaired hearing, dizziness and some nausea, but no vomiting. Fundi were normal, there was no nystagmus, and the general condition appeared excellent.

Discussion: This patient, with a prolonged history of middle ear suppuration, presents many interesting features. The history of long-standing middle ear suppuration was followed by an acute exacerbation of the chronic middle ear suppuration. In turn, there appeared evidence of an otitic sepsis, for which operation was performed. A few days later, labyrinthine symptoms appeared, progressive papilloedema, headaches, nausea and vomiting, and increased intracranial pressure. There was no localizing signs sufficient to warrant a diagnosis of a lesion of the cerebellum or temporal lobe. Ventriculography did not clarify the picture except insofar as it indicated no displacement or no dilatation of the ventricles or as commonly seen in hydrocephalus. This patient, on many admissions, has always had increased intracranial pressure, the initial pressure on lumbar puncture usually varying from 210 mm. to 260 mm. The fluid always was clear, with none or very few cells, and always the Pandy test was negative. Of interest has been the gradual subsidence of the papilloedema, so that one can now see relatively normal fundi; despite this, the patient still complains of occasional severe headaches. At present, therefore, one ought to regard this case as one of acute but "benign" otitic intracranial hypertension without obvious hydrocephalus.

49 East 96th Street.

OPERATION FOR THE CURE OF POSTAURICULAR FISTULAE. REPORT OF EIGHT CONSECUTIVE CASES.*

DR. SAMUEL D. GREENFIELD, Brooklyn.

Plastic surgery has rapidly gained ground as a distinct specialty in the field of general surgery. And justly so — for from the reconstructive aspect especially has it earned for itself the dignity and respect it justly commands from an appreciative medical public. Plastic surgery, generally speaking, should be relegated to those who because of inherent skill and special training are particularly suited to engage in its practice.

In the case of repair and cure of postauricular fistula, the subject with which this paper is mainly concerned, the plastic surgeon is confronted with a problem, for the ultimate solution of which he must look to the otologist. Owing to the fact that in conjunction with the performance of the plastic operation for the cure of postauricular fistula, the primary step is a complete and most meticulous revision of the original mastoid operation, it falls perforce to the lot of the otologist to acquire the knowledge and skill of the technique which constitutes the plastic phase of the operation. One must agree that this is a far easier solution to the problem than to make it compunctive for the plastic surgeon to acquire the necessary skill to practice mastoid surgery. It is for this reason that I believe this operation belongs and will remain in the domain of otologic surgery.

Most otologists with wide experience have in the course of their practices found cases in which the mastoid wound has failed to heal, and as a result there has become established a permanent postauricular fistula. Whatever may be their explanation, they recall in retrospect that the same care and assiduity have been exercised in the operative technique, particularly in regard to the thoroughness of the operation. Nevertheless, these wounds fail to show the usual reparative

*Presented before Clinical Society, Beth Moses Hospital, March, 1937.

Editor's Note: This ms. received in Laryngoscope Office and accepted for publication, June 14, 1939.

processes. Sutures slough, and primary healing does not take place. There is absence of filling-in of the mastoid defect by newly formed tissue, and the wound flaps become depressed and their skin edges inverted. The latter adhere to the underlying bone and leave a defect of variable size, usually in the region of the antrum.

This bony excavation becomes either partially covered with a thin epidermis or it remains widely open, with the presence of fine, unhealthy granulations, from which exudes a variable amount of mucoid discharge. In those cases where the epidermis attempts to close over the defect, it oftentimes does so only to the extent of leaving a small pin point opening, from which the discharge, although scanty in amount, continues to drain. Following head colds and upper respiratory infections, the delicate skin covering oftentimes quickly disintegrates and the defect assumes its original appearance. In those cases where there is no attempt at epidermization the fistulous cavity shows evidences of reinfection by an increase in the amount of discharge and inflammation of the adjacent tissues.

The condition of the tympanum is interesting. In the majority of cases the middle ear becomes dry and undergoes resolution, despite the fact that the antrum is patent posteriorly. The drum membrane is usually closed but does not assume entirely its normal characteristics. The landmarks are visible, but the drum membrane has only little or no lustre and remains definitely thickened. Hearing in most cases is only slightly affected, though in several instances there seemed to be more marked impairment.

What pathological basis exists for the establishment of a postauricular fistula does not seem to be very clear. It is a notable fact that this failure to heal occurs oftentimes in children who have good care, live in excellent hygienic surroundings, and are well fed and well nourished. It seems that under ordinary conditions these factors should be highly conducive toward increasing the reparative and healing functions of the body. Two of my cases occurred in children of physicians, where every known method was employed to facilitate and encourage healing. Nevertheless, these children developed large, persistent postauricular fistulae.

It is not within the province of this paper to offer any scientific explanation for the failure of these wounds to heal.

So far as the literature is concerned, one is struck by the absence of publications dealing with this most important subject. I am firmly convinced that the postoperative treatment of our mastoid wounds plays little or no part in determining whether or not a case is to develop a fistula. Most otologists will agree that mastoid wounds have been left widely open, in which primary union has not taken place. Nevertheless, with practically no care they have rapidly granulated and healed. On the other hand, a wound that has received every possible care and attention, with an effort to promote healing, has failed to do so. I, furthermore, cannot share the belief expressed by some otologists that the establishment of such a fistula is solely dependent upon the presence of some unexplored focus in the mastoid process. The untenable nature of this opinion must seem very evident when one realizes the fact that a thorough and complete dissection of the mastoid is not the prevailing technique in many instances, so that one would under these circumstances expect a far greater number of cases of postauricular fistula than do occur. It seems to me that there exists something in the form of a local devitalization of the tissues or some general dyscrasia which is responsible for retarding and arresting the normal healing and reparative functions.

There are, however, a group of cases which do not fall into this category and to which I wish to refer briefly. They also present fistulae, but not of the type under discussion. They are cases of acute mastoiditis in which complete closure of the wound is arrested, or in which after being closed for a short period again reopens. These so-called fistulae are small and present very little or no bony defect. They are as a rule filled-in with an abundance of granulations. The mastoid scar appears slightly edematous and gives one the impression that there is something underneath the tissues. In these cases, sooner or later, a sequestrum presents itself in the fistulous opening; or upon probing and investigation of the wound one discloses its presence. Invariably after removal of the sequestrum the fistulous opening heals rapidly.

In the operative treatment and cure of postauricular fistula, certain fundamentals must be borne in mind. In a well established postauricular defect there have been created two distinct pathological conditions which are responsible for the

persistence of the fistula. First of all, there is present after some duration of time a definite bone focus which must be completely eliminated if we are to hope for a cure. The type, character and process of development of this focus I shall later describe in detail. Second, the skin and soft tissues in and about the fistulous opening become atrophied and of such devitalized nature that they are unsuitable for the purpose of building up and covering over the defect. It is necessary, therefore, in order to obtain firm union and at the same time a good cosmetic result, to bring new tissues to the part in order to accomplish these ends.

Several years ago, before giving this problem serious thought, I attempted the closure of a number of fistulae with various methods. There were two cases in which the adherent skin about the fistulous opening was elevated from the bone and a portion of the pale thin scar excised and the edges sutured together. No attempt was made to either open or clean out the mastoid. The result in these two cases was a total failure. There was then a third case, in which I followed the technique suggested by Straatsma and Peer.[1] They reported excellent results with the use of fat tissue implants. In this patient, as in the two preceding cases, I did not clean out the mastoid; however, I made a posterior incision and a sliding flap to prevent as much as possible any tension upon the sutures. The fat implant soon became infected and the wound broke down completely. In a fourth case it occurred to me that perhaps the mastoid itself should be revised. This was a young boy, age 11 years. He had a large, deep postauricular fistula, in which there had been one previous attempt at repair. I was, indeed, surprised to find the degree and extent of bone involvement in this mastoid. I did a very thorough revision of the latter. A large portion of the tip was removed in order to permit the digastric muscle to present itself into the cavity. The posterior canal wall was lowered considerably in order to diminish the depth of the defect. A posterior incision was not made. The scar was excised and the wound sutured in the usual manner. This case ultimately healed up, but only after many weeks of patient and persistent treatment. There was no attempt at filling-in. The defect remained very pronounced but was completely epidermized. Cosmetically, it was, indeed, a very poor result. I had a second case in a child, age 6 years, in which

there had been two previous attempts at closure. The defect in this child was unusually large. I reoperated the mastoid and found a diseased tract of cells extending far forward in the zygoma. I lowered the posterior canal wall and removed the newly formed tip well above the digastric ridge, so that a considerable portion of the digrastric muscle presented itself into the cavity. A posterior incision and sliding flap were made. The skin of the fistulous tract was excised and the edges brought together. This case healed more rapidly than

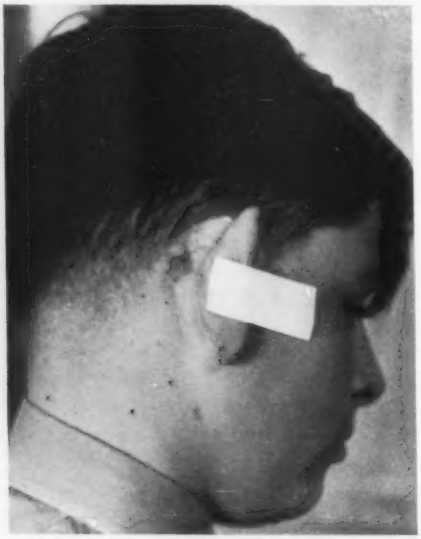


Fig. 1. Small pin point fistula with epidermization, intermittent discharge following head colds. Duration of fistula, seven years.

the preceding one, but also took considerable time because of the enormous bony defect. Here, again, the functional result was good, but from the cosmetic aspect it was poor. These two cases, when viewed in the light of the preceding three, confirmed my opinion that the reason for failure in the latter was due to the fact that I had omitted that very essential step of cleaning out the mastoid. I believe, therefore, that the eradication of this newly formed bone focus is of prime importance. That such a bone focus exists is most certain from

my observations in these eight consecutive cases. Opening of the mastoid and careful investigation in each one disclosed the presence of definite tracts of infected cells in a number of locations. Favorite sites for these regenerated groups of cells are, in order of frequency, the zygoma, the Citelli angle, the floor of the middle fossa, the periantral region and along the posterior canal wall into the tip. I believe these cells are newly formed structures that have developed subsequent to the original mastoid operation and are not residua that the

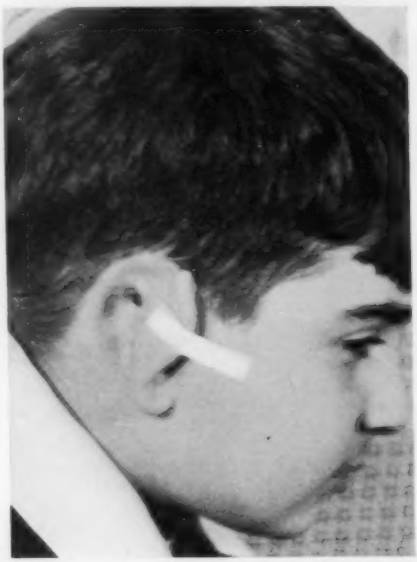


Fig. 2. Small fistula with fine granulations. Discharge constantly present. Duration of fistula, two years.

surgeon has failed to eradicate. It is a well established fact that new cells can and do reform after mastoid operations. Not alone do new cells develop, but the internal table often regenerates to the extent of assuming the characteristics and anatomical conformation of normal plate. With the return of these bony structural relations, the exposed bone cells become infected from without and a well established low grade osteomyelitis develops. As a result of this slow process of bony necrosis there is formed overhang, diseased cell tracts

and pockets of various kinds. Along with this prolonged bony disintegration there follows concomitantly the formation of diseased granulation tissue.

It can readily be seen, therefore that in a given case, which at the time of the original mastoid operation possesses some local or general dyscrasia responsible for the failure to heal, may later on, even though the dyscrasia disappears, fail to respond because of the establishment in the interim of this new bone focus. It is, therefore, essential as a first step in

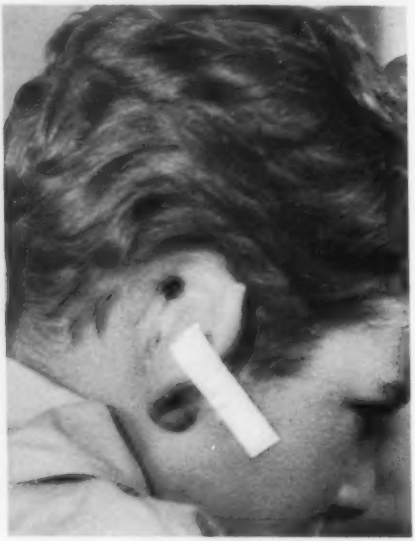


Fig. 3. Moderately large sized fistula. Granulations abundant, with constant discharge. Duration of fistula, four years.

the cure of postauricular fistula to completely and thoroughly eradicate all vestiges of diseased bone and granulations.

Another concern which is of great importance to the patient is the cosmetic result. The surgeon should, therefore, not lose sight of this fact in his effort to repair the fistula. It is in this regard that the otologist must bear in mind that he assumes the responsibility of the plastic surgeon when he undertakes to step into his domain. The defect must, therefore, be filled-in and this can only be accomplished by bringing

new tissue into the cavity. Eagleton[2] first suggested used bone chips with which to fill in the bony defect. Others suggested fascia lata, fat implants and muscle tissue. The most practical and useful is the temporal muscle, which is easily accessible and can be reached by simply extending the mastoid incision. It serves to bolster up the wound and adds vitality to the overlying undernourished skin, supplying it with additional circulation and thereby insuring perfect union. The eight cases reported have been operated according to the

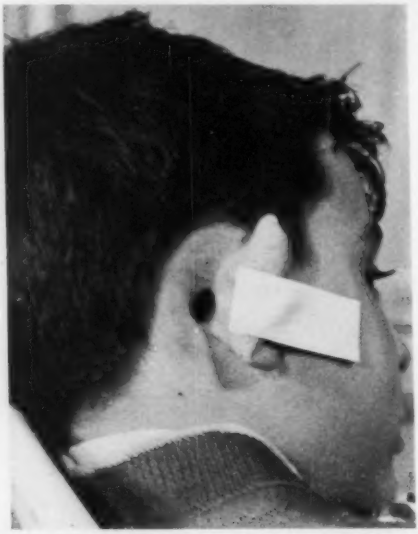


Fig. 4. Large size postauricular defect, with constant thin discharge. Duration of fistula, six years.

technique that follows. A complete cure was obtained in all of them. Both functionally and cosmetically, the end-results have yielded all that can be desired.

The operation here described for the cure of postauricular fistula presents some features to which I do not wish to claim originality. The posterior incision and sliding flap for the prevention of tension upon the sutures has been used for some time. Likewise, the transplant of various tissues into the bony defect, as I have previously mentioned, has been

employed before. Opening of the mastoid has also been done to some degree, but the necessity of being most scrupulous when searching for and cleaning out all the diseased tracts has not been stressed sufficiently. I wish to emphasize again that this is the most important phase of the entire operation. Many cases will eventually heal with the employment of this alone. But to insure more rapid and permanent recovery, and especially to obtain a good cosmetic result, several additional features have been inaugurated. The size of the bony defect

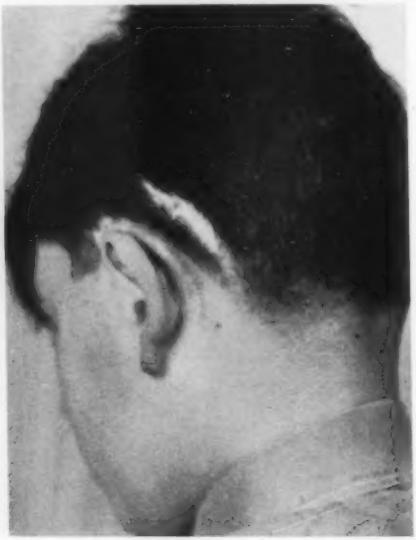


Fig. 5. Case 1.

is diminished by lowering the posterior canal wall and by wide removal of the mastoid tip and the beveling off of all surrounding bone. The exposure and bringing up of the digastric muscle has not been employed heretofore. The method of handling the temporal muscle flap, and especially the technique for suturing the incisions are some of the innovations which have added materially in insuring a satisfactory result. Above all, it is the combination of all these details that will greatly minimize the possibility of failure. I do not doubt that occasionally a case may clear up without the use of what

appears to be one or two of the so-called minor details. But I am convinced that with the method to be described there will be a maximum assurance of success.

OPERATION.

1. Revision of the Mastoid Cavity: The incision is made through the old scar and carried down to the bone. In the elevation of the soft tissues, an effort should be made to take along with them the periosteum. Great care should be exer-

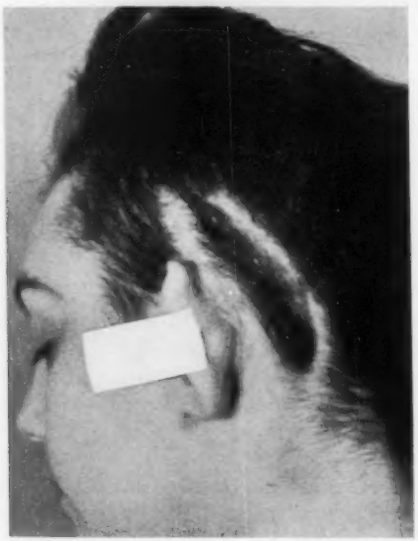


Fig. 6. Case 7.

cised not to traumatize unnecessarily the already devitalized tissues. The anterior flap should be pushed far forward in order to well expose the entire posterior canal wall and the zygomatic region. The posterior flap is then elevated, with its attached periosteum, for only a short distance posteriorly.

The mastoid is now carefully and completely revised. All diseased tracts are to be looked for and thoroughly cleaned out. The tip should be removed well above the digastric ridge so as to permit the digastric muscle to present itself into the

cavity. The posterior canal is lowered considerably and, wherever possible, the surrounding bony structures, both superiorly and posteriorly, should be beveled down with the large gouge so as to diminish the size and depth of the original bony defect. All diseased granulations are carefully removed from the antrum, and the horizontal canal is clearly visualized.

2. Sliding Flap for Relief of Tension of the Sutures: This incision is made parallel to and about one and one-half to two inches posterior to that of the mastoid incision. The anterior flap is elevated, with its periosteum. This is best accomplished by working the elevator from behind forward until the latter presents itself into the mastoid cavity. The flap should be freed entirely from the posterior aspect of the tip and the lower portion of the adjacent occipital bone, where it is usually firmly adherent. This is very necessary if the flap is to slide forward easily to relieve the tension of the sutures at the lower end of the mastoid wound. All bleeding is controlled and the area covered and protected with gauze moistened in saline.

3. Preparation of Temporal Muscle Flap: The original mastoid incision is carried up over the temporal region for a distance of two inches or more. The skin is dissected away and the muscle exposed. The bleeding, which is usually profuse and troublesome in this region, must be completely controlled. After the muscle tissue is taken away from the temporal bone, a potential cavity is created and, with incomplete hemostasis, blood is apt to collect in this space after application of the dressing. We know the readiness with which infection takes place under these circumstances so that every precaution must be taken to avoid such a complication.

The muscle is lifted off the underlying bone and a wide, fan-shaped flap is cut with the pedicle attached anteriorly. The flap is then pulled down into the mastoid cavity. If it does not reach adequately into the defect, the upper anterior attachment of the muscle fibres are severed a little at a time to allow the latter to be pulled down into the desired position. Care must be exercised not to thin down the pedicle too much for fear of cutting off its blood supply. When the muscle flap is placed into the cavity it is held in this position by suturing to the tissues in the lower end of the wound. It is also advis-

able, wherever possible, to bring some digastric muscle fibres up into the cavity. Occasionally the two muscles can be sutured together. This latter procedure, I believe, offers the best assurance so far as the vitality of the tissues is concerned and greatly favors firm and permanent union.

TABLE I.

Case	Sex	Age	Duration of Fistula	Previous Attempts at Closure	Previous Mastoid Operations	Impairment of Hearing
1	Male	11	6 years	None	2	Marked
2	Male	5	3½ years	One	2	Slight
3	Female	4	8 months	None	1	Slight
4	Female	7	2½ years	None	2	Slight
5	Female	10	4 years	None	1	Moderate
6	Female	8	3 years	None	2	Marked
7	Female	17	8 years	None	1	Moderate
8	Female	9	4 years	One	3	Moderate

TABLE II.

Condition of Middle Ear	Location of Diseased Cells at Operation	Flap Muscle	Complications	Result
Dry	Zygoma and floor of middle fossa.	Temporal muscle	None	Cured
Dry	Citelli angle and along the posterior canal wall.	Temporal muscle	None	Cured
Moist	A well developed tract of cells in the zygoma.	Temporal muscle	Erysipelas	Cured
Dry	Zygoma and Citelli angle floor of middle fossa.	Temporal muscle	None	Cured
Dry	Zygoma and periantral region.	Temporal muscle	None	Cured
Moist	Zygoma and Citelli angle.	Temporal and digastric muscles	None	Cured
Dry	Zygoma and Citelli angle and along posterior canal wall.	Temporal and digastric muscles	Infected hematoma	Cured
Moist	Zygoma, periantral region, along posterior canal wall into tip.	Temporal and digastric muscles	None	Cured

4. Treatment of the Old Scar: We now return to the old scar and carefully excise the tissues about the old fistulous tract. The edges of the scar throughout its entire length are trimmed away. This allows for better coaptation of the skin and underlying tissues. Care should be exercised, however, not to sacrifice too much of the scar tissue.

5. Preparation of Flaps for Suturing: The mastoid incision is the one in which we are especially interested to obtain primary union. In view of the fact that the tissues are more or less undernourished, it is important to insure healing by bringing them together with two layers of sutures. This can be facilitated by carefully dividing the tissues with sharp dissection into a superficial and deeper layer for a short distance in order to permit the needle to pick up the deeper tissues. Catgut sutures are placed fairly close together along that part of the incision immediately over the original bony defect. The remainder of the incision may be closed with only one layer of sutures. It will be noted that as a result of bringing the deeper tissues firmly together there is an overabundance of skin which now approximates very easily. This skin immediately over the bony defect is held together by applying skin clips. Care should be taken not to make too much pressure with the clips in order to avoid sloughing and necrosis.

6. Suture of Posterior Wound: The posterior incision is closed with interrupted silkworm gut sutures. If bringing the edges together causes undue tension upon the mastoid sutures, it is better to forego perfect coaptation and be content with a small amount of gaping. This incision granulates rapidly. Furthermore, the scar is well within the hair line and is not visible after the hair returns.

7. Insertion of Drains: A small, narrow rubber drain is inserted into the lower end of the wound, reaching into the mastoid cavity for a short distance. A small drain may also be placed into the upper angle of the incision over the squama and directed forward into the area where the temporal muscle flap has been taken.

8. Dressing: Firm pressure is made over the operated field in order to express any blood that may have collected. A layer of vaseline gauze is placed over the entire wound in order to facilitate the removal of the dressing subsequently. To avoid the collection of blood and exudate, two small rolls of bandage are placed, one over the temporal region and one over the posterior flap. To these, firm pressure is applied with the gauze dressing and the bandage.

9. Postoperative Treatment: The postoperative reaction as a rule is slight. The temperature may rise to 101° or more the next 24 hours, but if no complication supervenes it

becomes normal within 72 hours. The usual postoperative pain in the operated area is complained of, and inability to open the mouth widely results from the surgical trauma of the temporal muscle.

The first full dressing is performed on the third day, and clips and sutures are removed on the fifth day. As a rule primary union takes place and the drains are removed and left out after the second dressing. The bandage comes off in the third week, and the wounds are completely healed by the end of the fourth week.

The eight cases operated according to the described technique have all recovered and the cosmetic results have been most gratifying. What appeared as an unsightly disfigurement has now been completely eliminated. The discharging fistula is replaced by firm, healthy tissue. The scar in the squama and that of the sliding flap, being inside the hair line, are both hidden from view.

In those cases in which the hearing was impaired, a noticeable improvement has been observed after the operation. This has been further corroborated by audiometric hearing.

COMPLICATIONS.

There were no serious complications in the cases reported. In one case, preparation of the sliding flap required rather extensive separation of the tissues from previously exposed sinus and dura. In this patient, a wide subtemporal decompression had been done eight years before for an otitic meningitis. In this case, Case 7, there resulted postoperative bleeding beneath the tissues in the temporal region, with subsequent infection of the upper end of the original incision, and the patient made an uneventful recovery. Case 4 developed an erysipelas. She ran a rather severe course but recovered, with a fine cosmetic result.

REFERENCES.

1. Straatsma, C. R., and Peer, L. A.: *Arch. Otolaryngol.*, p. 620, April., 1932.

2. Eagleton, W. P.: The Reconstruction of the Mastoid Wound Cavity by the Use of Bone Grafts and Chips. The Laryngoscope, 29:272, May, 1919.

169 New York Avenue.

AUDIOMETRIC RANGE IN ALLERGY.*

DR. GLENN J. GREENWOOD, Chicago.

Reports of allergic reactions involving the external and inner ear are quite numerous, but to date allergic states of the middle ear which clinically may be present have not been borne out by cytological and pathological investigations.

Grahe writes that concerning allergic disease of the ear only hypothetical statements may be made.

Piness states that vasomotor congestion as seen in the epipharynx may extend up the Eustachian tube, invading the middle ear.

Lewis reports six cases of acute otitis media he thought of allergic etiology, but no cytological examination confirmed his findings.

Proetz, writing relative to allergy in the middle and external ear, feels that localized allergic reactions may simulate other ear diseases by the mechanical reaction of edema upon the tissues of the tympanum.

Hansel states that involvement of the middle ear with an allergic lesion comparable to that seen in the nose characterized by edema, eosinophilic infiltration and eosinophiles in the discharge, has as yet not been reported. Chronic otorrhea, he writes, may show some eosinophiles, and aural polypi, too, may be infiltrated with them, but their presence is due to prolonged irritation as seen in chronic appendicitis and tumors, such as carcinoma.

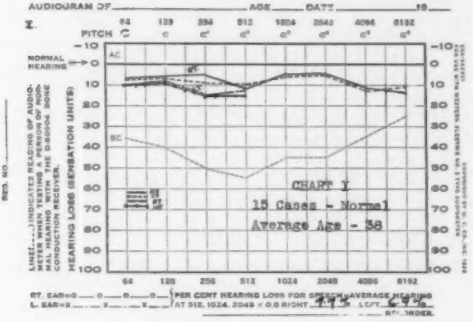
Examinations of aural secretions also have never demonstrated excessive numbers of eosinophiles present, in either acute or chronic otorrhea occurring in allergic individuals.

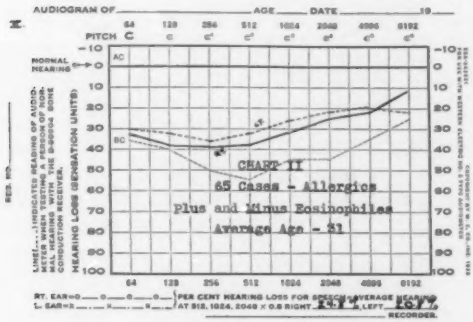
Kerrison states that the mucous membrane lining the tympanic cavity is directly continuous through the Eustachian

*From the Ear, Nose and Throat Department of Northwestern University.
Editor's Note: This ms. received in Laryngoscope Office and accepted for publication, June 14, 1939.

tube with that of the nasopharynx. The nasal mucosa, responding to allergens as it does with edema and eosinophiles, the question has arisen whether there may not be an impairment of hearing, simulating the conductive type, resulting from a water-logging of the mucosa of the middle ear cavity.

Accordingly, 65 unselected allergic individuals had nose,

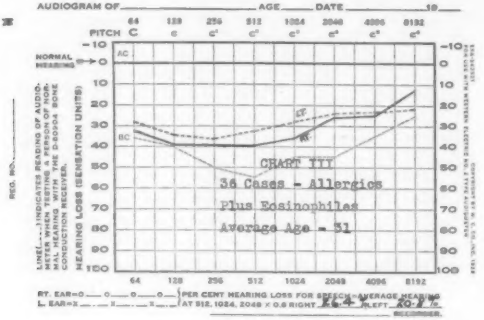


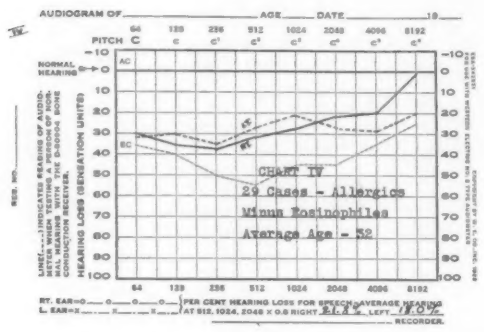


throat and ear examinations. Their hearing was tested with 2A and 3A Western Electric Audiometers. Nasal smears were taken in all cases. Fifteen persons with normal hearing were used as controls. There were 35 females and 30 males, the youngest was age 11 years; the oldest, age 71 years; the average being 31 years. Forty-one gave a history of seasonal, perennial or both types of hay fever. In one instance, a low

tinnitus accompanied the attack. Thirty-five admitted asthmatic attacks, 11 of these also manifesting nasal allergy; seven had urticaria; two, purpura. One had abdominal pain on eating butter; seven had joint pains; one had acute rheumatic fever.

Relative to the ear, none complained of stuffiness; six stated they possessed impaired hearing, and one was definitely





worse during attacks; four had tinnitus, while five gave a history of rotatory vertiginous attacks.

Thirty-six of the 65 cases presented excessive numbers of eosinophiles in nasal smears, while in the remaining 29, eosinophiles in greater than normal numbers were not present. The appearance of the nasal mucosa of the allergic rhinitis group varied, not all exhibiting the pallor thought by

many to be ever-present. Twenty-six presented evidence of bilateral maxillary sinus pathology, 17 of these being in the allergic rhinitis eosinophilic group. Only one frontal sinus was involved.

The nasopharnyx, as viewed with the nasopharyngoscope, showed polypi in four cases, pharyngeal tonsil hypertrophy in five; the sphenoidal fissure was hyperemic in one, and contained pus in two subjects. Neither of these cases presented thickening of the plica septi. The balance of the nasopharynx presented its characteristic pale mucosa. The pharyngeal opening of the Eustachian tubes was in no instance encroached upon by any nasopharyngeal mass.

The tympanic membrane showed changes in only 13 cases, ranging from a dusky peripheral hyperemia in a retracted drum to a thickened, opaque, normally-placed membrane. No tympanic scars, chalk plaques or dehiscences were noted.

Using the pitch range audiometer, the ear with the greater acuity being tested first, the recordings were started with the 512 d.v. (C_2), descending the scale to the 64 d.v., then again beginning at the starting point and ascending the scale to C_6, or 8,192 d.v. per second. The rationale of this procedure is that the pitches ranging from 512 to 2,048 are the basic frequencies essential to speech, consequently most often perceived. It is upon the average reading here obtained that the per cent of hearing loss for speech is calculated.

In addition to testing the normal cases exactly as the allergic subjects were, the readings from 64 to 512 were recorded, ascending the scale. This produced an interesting factor, by showing a greater hearing acuity ascending from 64 to 512 than descending from 512 to 64, there being a discrepancy at 512 of 6 dcb. in the right, and 3 dcb. in the left ear. This factor was attributed to the "warming up process," which is a basic physiological neuromuscular phenomenon, and demonstrates the fact that the ability of the same subject to respond varies.

It is the impression that the correction of this variable can be facilitated by first permitting these patients to have a given period of rest preceding their tests, such as is now used in B.M.R. determinations; and, second, allowing a trial reading before plotting the final audiogram.

Table I rather bears out the statements made that the average hearing of the normal individual is 8 to 10 dcb. below the line indicating normal hearing as plotted upon the audiogram chart.

TABLE I.

Type of Case	Number of Cases	Average Age	Per Cent Hearing Loss for Speech (Average Hearing at 512, 1,024, 2,048 x 0.8) Rt. Ear	Lt. Ear
(A) All allergics	65	31	24.8% (A-D=16.5%)	20.8% (A-D=14.1%)
(B) Allergics with eosinophiles	36	31	26.4%	20.8%
(C) Allergics without eosinophiles	29	32	21.8% (B-C=4.6%	18.0% (B-C=2.8%)
(D) Normals	15	38	8.3%	6.7%

The allergic cases showed a marked deficit in the lower tone limit as compared to the normals, while the upper tones tended to approach each other more equally.

The per cent hearing loss for speech of the allergic cases was 16.5 per cent greater in the right ear, and 15.2 per cent more in the left ear than in the normal readings. The average age of the allergic patients was seven years younger than the normals.

In conclusion, may it be said that these findings are merely based on audiometric records, that the more accurate tuning fork tests have, for the most part, been omitted, and that no sections of tympanic mucosa have been obtained nor cytological examinations made. Until the latter is done, it is believed that the foregoing findings must, of necessity, be accepted with reservations.

55 East Washington Street.

SUFFOCATION DUE TO ALCOHOL INTOXICATION.

DR. CAESAR HIRSCH, New York.

If we look through the medico-legal, pathological and laryngological literature we frequently find mentioned the fact that death may occur in drunken persons if foreign bodies, as, for instance, vomited stomach contents, are drawn into the opening of the larynx; however, there is no case history to be found in the entire literature, with the exception of one, mentioned by Chevalier Jackson.[1]

While working in a hospital, Jackson was called into an adjoining operating room, where a surgeon and his assistants had tried forced mask respiration, then tracheal intubation by blind method. The mask method had given relief for a time but the patient had gradually become unconscious and cyanotic. The surgeon's assistant was an expert in blind intubation and could not understand his inability to intubate in this instance. Jackson took with him his laryngoscope, and exposure of the larynx revealed occlusion with a grayish mass, which proved to be meat. Intratracheal oxygen insufflation after removal of the meat kept the man alive until he could be trusted to do his own breathing.

The patient was in a state of profound alcoholism when brought in from the gutter in front of the hospital and doubtless the meat had been vomited.

That the respirator machine had forced the meat further into the larynx is no criticism against the machine for the general run of cases. Had the laryngoscopist not been available, the diagnosis of the vomited piece of meat in the larynx could not have been made and the patient might have died, and without a postmortem examination the real cause of the death in this case would not have been established. Even if the surgeon had done a tracheotomy, the patient's life might have been saved, but the diagnosis of a vomited piece of meat drawn into the larynx could not have been made. If the patient had regained consciousness and breathed through the

Editor's Note: This ms. received in Laryngoscope Office and accepted for publication, May 23, 1939.

tracheal tube, he either might have again swallowed the meat from his larynx, or might have expectorated it later without anybody's knowledge.

It may, therefore, be of interest to report the following case:

R. A., male, age 45 years, died suddenly in May, 1937. He had been married 18 years at the time of his death. In his business he was compelled to entertain quite frequently and to drink with his customers. During his lifetime, he was frequently intoxicated.

He had an automobile accident in 1924, at which time he complained of double vision and sleepiness. This condition lasted about three months and never recurred.

On the day of his death he played 18 holes of golf, ate dinner, danced, drank quite a good deal and was put on a cot to rest. One of the employees of the club noticed him vomiting and then struggling for his breath; his face became blue and then black. A doctor was called to render first aid but found the man already dead, and signed the death certificate, giving the cause of death: "Acute dilatation of the heart caused by strangulation, due to the swelling of the posterior part of the tongue."

Since the widow put in a claim for double indemnity, an exhumation of the body was made.

Autopsy was performed by Dr. John Kolmer and Dr. Theodore J. Curphey on Sept. 28, 1937. The following is a brief abstract of the report:

The body was properly identified; it showed external signs of decomposition, but the brain and internal organs were very well preserved and satisfactory for thorough examination. Insofar as could be determined, there were absolutely no evidences of injuries, fracture, and so forth.

Organs of the Neck: The tongue, epiglottis, larynx, trachea and upper portion of the esophagus were moved *in toto*. The mouth was found to be edentulous. Lying between the gum and the buccal mucosa, on the left side, were seen particles of food material — amounting in total volume to about the size of an English walnut. Two types of structure were recognizable:

1. A soft, green, herb-like material having a faint aromatic odor.

2. Flat sheets of glistening fibrillar yellow-white structure, suggesting elastic tissue of meat.

The tongue was rather large. Scattered on its posterior surface and on the foramen cecum were seen similar but smaller amounts of food material. Inspection of the larynx showed sufficient food material of a similar type to almost completely block the airway. The material lay loose in the lumen of the larynx and was entirely above the level of the vocal cords. Careful search of the airway of the lower larynx and trachea showed no evidence of foreign material. The mucous membranes of the larynx were pale and showed no evidence of hemorrhage.

Examination of the *brain* and *meninges* revealed no hemorrhages on the surface or within the brain. Microscopical examination of the brain tissue revealed moderate edema of the meninges. There was associated moderate round-cell infiltration; the blood vessels were well preserved; the meninges of the cerebellum showed moderate number of round cells, many of which lay in the region of the blood vessels.

The *lungs, pleurae* and *bronchi* were essentially normal in gross appearance, with no food or foreign bodies in trachea or bronchi. Microscopical examination showed some passive congestion of the posterior lobes and some evidence indicating very minute capillary hemorrhages, probably resulting from violent coughing or suffocation.

The *heart* was grossly about normal in size but showed some increased thickness of the left ventricle. The mitral valve showed small, fibrotic verrucose lesions. Small fibrotic scars were found in the upper part of the left ventricle. Both anterior and posterior *coronary arteries* showed an extreme degree of sclerosis but there were no evidences of thrombosis or recent infarction. Microscopic examination showed a diffuse fibrosis of the heart muscle extending from markedly fibrotic (sclerotic) coronary arteries.

The arch of the aorta, along with its thoracic and upper abdominal portions, revealed an extreme degree of syphilitic

aortitis and atheroma along with the typical microscopical changes of syphilitic aortitis, producing partial occlusion of the ostia of both coronary arteries. Portions of the aorta were slightly dilated but there was no aneurysm.

The *liver* was normal in size but showed fatty infiltration upon microscopical examination.

Both *kidneys* were slightly enlarged and showed definite dilatation of the pelvis of each with dilatation of the uppermost portions of the ureters, especially on the left side.

The *stomach* contained some food material but was otherwise normal. Owing to the elapse of over four months since death, it was impossible to express any opinion on the presence or absence of alcohol.

A report of a physician who had treated the patient from March 23, 1932, to March 17, 1933, for cerebral syphilis with epileptiform seizures shows that the patient was treated with phenobarbital, mercury and iodides by mouth, and bismuth intramuscularly.

Laboratory findings during this treatment were as follows: Urinalysis: No pathology. Blood count, March 23, 1932: Hgb., 90 per cent; R.B.C., 5,150,000; W.B.C., 10,100; diff. polys., 60 per cent; basoph., 2 per cent; lymph., 38 per cent; R.B.C., normal.

March 23, 1932: Kahn test (blood), 1+. Calcium test, 11.2 mg. Sugar test, 118 mg.

April 8, 1933: Kahn test (blood), negative. Wassermann, negative.

March 29, 1932: Spinal fluid study: Acetone negative, and cholesterin negative with 0.2 cc. Acetone weakly positive with 1 cc., and cholesterin medium positive with 1 cc.

Cells: 12 cells and two units of protein. Colloidal gold: 4,444,442,100.

At the hearing before the Supreme Court the plaintiff proved that R. A. attended to his business daily; that he had an excellent memory; was quick, full of pep, energetic, always on the go, and played golf at least five times per week; that

he had never been confined to his bed even for a single day; that he had no medical treatment from March, 1933, to the day of his death, four years later in May, 1937; that at no time during these four years was there a rash on any part of his body, nor did he ever suffer from any epileptic attacks, nor did he exhibit any tremors, twitches or any other physical sign indicating illness or physical incapacity.

Numerous witnesses testified to the above, both business acquaintances and friends.

The insurance company contended as follows: That a combination of heart and brain disease caused the death, and that the patient had many attacks of similar character previously.

A bartender testified that he saw the patient have three attacks, during two of which he was drunk, and at which time he slumped over a chair.

An osteopath testified that he saw an attack, at which time he was drunk, and the only symptom exhibited was paleness of face. Another doctor testified that he saw him unconscious, but at that time he was not drinking. Three months before the death of the insured, the doctor, who testified that he lost his records, remembered being called in to treat R. A. and that R. A. had a high blood pressure, rapid pulse and that he was pale. At that time patient was intoxicated.

Stories were also told by witnesses that Mrs. R. A. had stated to them that once, while driving an automobile, R. A. began coughing, lost his breath and became unconscious; that he had been drunk at that time. On another occasion, about eight years before, while dancing, he fell over and his false teeth slipped out of his mouth; that he became pale, and a doctor gave him first aid; that another attack occurred while he was driving an automobile, and that a physician gave him first aid. All of these last three or four attacks were contradicted by witnesses that such had ever occurred.

In substance, such were the contentions of both parties.

There was testimony that it was an inactive syphilis, on behalf of the plaintiff, whereas the defendant claimed that the syphilis was active at the time of the death of R. A.

The jury decided in favor of the plaintiff; *i.e.*, that death was caused by suffocation, caused by a vomited piece of meat drawn into the larynx in a state of alcohol intoxication.

During the examination and cross-examination, the expert was asked various questions by the respective lawyers, of which the following may be of interest to be mentioned:

Question: Is it possible that the deceased died from a drawing in of a piece of meat into the larynx, even though there was no evidence of hemorrhage in the mucous membranes of the larynx, and even though the section of the soft tissues above the larynx, such as the thyroid and the adjacent muscles, did not show any evidence of hemorrhage?

The answer was in the affirmative by quoting Gonzales,[2] who states, on p. 276: "The larynx in cases of smothering may not show any cyanosis or submucous hemorrhage."

Another question was: How did it come that R. A. could have died from suffocation by the piece of meat drawn into the larynx, although — according to the autopsy — the food material was lying entirely above the level of the vocal cords, and careful search of the airway of the lower larynx and trachea did not show any evidence of foreign material?

The answer was: In such cases sudden death may occur by a laryngeal shock. Von Hofmann and Haberda,[3] on p. 683, mention cases in which the patients collapse in the moment of the drawing in of a bite into the larynx as if stricken by lightning, without showing any dyspneic or clonic symptoms.

It is well known that sudden stop of the cardiac and respiratory action may occur under the influence of a mechanical irritation of the larynx.

Hofmann and Haberda also mention sudden suffocation caused by a dental plate, which, while the person was sleeping, was drawn into the larynx by aspiration. Postmortem examination in this case did not show any lesion of the larynx nor any hemorrhages in the deeper airways (trachea, bronchi or alveolar spaces).

Another question was: Are there not many cases in which at the postmortem examination foodstuffs regurgitated from the stomach are found in the throat?

The medical expert replied that it was difficult to anwer this question since "throat" was too general a term.

The next question of the lawyer was: In the pharynx or in the larynx?

The answer was: In a great number of cases in the pharynx, but never in the larynx if the person did not die from suffocation.

Another question was: Why is it that this suffocation due to aspiration of vomited foodstuffs into the larynx happens so often to alcoholics and not to syphilitics?

The answer was: The reflex deglutition is disturbed by the intoxication with alcohol and thus fails to work smoothly, because of the relaxation of the normal vigilance of the protective reflexes. Our common experience with alcoholics tells us that intoxicated persons often have vomiting spells, accompanied by aspiration of foodstuffs into the larynx. If the intoxication is not too severe, these people expectorate the foreign bodies from the larynx, or even the subglottic space and trachea, by severe coughing.

Syphilitics do not show this type of disturbance of the reflex deglutition. If there is a disturbance of the deglutition reflex due to syphilis, it is always caused by an affection of the medulla; however, this kind of disturbance of the larynx is accompanied by dysphonia and difficulty in swallowing as so-called bulbar symptoms of syphilitic meningitis or gummata in the medulla in the area of the nuclei of the vagus (pneumogastric) glossopharyngeal and hypoglossal nerves; but this condition is never an acute one and is accompanied by so many other symptoms that it could not have been the case in this particular instance.

This fact leads us to the very important question of the cross-examining lawyer: How can you state that the death in our case was not caused by the cerebral syphilis with epileptiform seizures, for which the man was treated, or by the chronic leptomeningitis, probably luetic in nature, found in the autopsy?

The answer to this question was: R. A. was treated for cerebral syphilis from March 23, 1932, until March 17, 1933. Until the time of his death in May, 1937 — that means for more than four years — he was not ill, and was not compelled to see a physician for any kind of treatment. If, at the time

of his death, he had suffered from a cerebral syphilis, for which he was treated in 1932, or from a chronic luetic leptomeningitis, as was found in the autopsy, the man necessarily would have shown so many other symptoms that he would not have been able to play 18 holes of golf on the day of his death, nor to dance and speak with so many people as he really did. He did not complain about headaches, insomnia, dizziness, attacks of unconsciousness, failure of memory, eye disturbances, nor did he show any other symptoms which would have been observed by his associates.

It is without any question that the deceased did not have to be treated during the last four years of his life, and the very scant findings of the chronic leptomeningitis, probably luetic in nature — as, for instance, the moderate edema of the meninges and the moderate round-cell infiltration, without any affection of the blood vessels — show that at the time of his death the syphilis of R. A. was arrested.

And so another question of the cross-examining lawyer — Do you not know that syphilis and alcohol are sometimes very good friends? — could be answered in the affirmative — that oftentimes syphilis may be acquired when too much alcohol was imbibed; but in our case the alcohol, in all probability, was alone the cause of the vomiting and suffocation, because R. A. did not show any symptoms of twitchings of the facial muscles or limbs, of jerks, or convulsions, which would have been noted by the bartender or the other employees of the club who saw him vomiting and struggling for breath.

People suffering from epilepsy or epileptic attacks due to syphilitic meningitis have convulsions, or at least slight jerks of the head, but are at the time of such an attack unconscious and, therefore, do not struggle for breath.

It may be interesting in this connection to quote such experienced authorities as Chevalier Jackson and Chevalier L. Jackson,[4] who state, on p. 132: "Obstructive foreign bodies may be quickly fatal by impaction in the glottis on inspiration, or in the conus elasticus on abortive becchic expulsion. The authors have hundreds of newspaper clippings giving accounts of death of patients before the arrival of a physician. It has happened in our experience, as in that of many other sur-

geons, that an emergency tracheotomy had to be done with a pocket knife to save life. The absence of convulsions differentiates obstructive asphyxia from the epileptic seizure, and the obstructive nature of the asphyxia is indicated by the in-drawing at the suprasternal notch, the epigastrium and the intercostal spaces. There is a fight for air in impending asphyxia, but during the fight the patient is conscious; in an epileptic seizure the patient is unconscious during the convulsions. In asphyxia the patient is limp and blue, then unconsciousness supervenes."

The authors show a drawing of an autopsic specimen, showing an oyster at the bifurcation of the trachea. The case was that of a man, age 60 years, who, while eating raw oysters, suddenly choked, struggled for air, became unconscious and died within 7 minutes.

In such a case, of course, even tracheotomy would not have been able to save the patient's life.

Besides that, we learn from literature, as, for instance, MacCallum[5] mentions on p. 415, that it is not uncommon to find in the autopsy of drunken persons who have died with symptoms of choking, masses of meat so firmly wedged into the glottis as to be removed only with difficulty.

We do not find, however, in the entire pathological[6, 7] and medico-legal[8, 9, 10] literature any cases of suffocation caused by aspiration of vomited material due to meningeal or cerebral syphilis.

SUMMARY.

A case of suffocation caused by aspiration of a vomited piece of meat is reported.

The question whether death was caused by aspiration of a foreign body in a state of alcoholic intoxication or by a previous cerebral syphilis or luetic leptomeningitis is discussed, and the conclusion is made that death in this case was due to alcoholism.

BIBLIOGRAPHY.

1. JACKSON, CHEVALIER: Peroral Endoscopy and Laryngeal Surgery (Textbook), p. 75, 1915.

2. GONZALES, THOMAS ARTHUR; VANCE, BENJAMIN MORGAN, and HALPERN, M.: Legal Medicine and Toxicology. New York and London, 1937.

3. VON HOFMANN, EDUARD; and HABERDA, ALBIN, and VON WAGNER-JAUREGG, JULIUS: Lehrbuch der Gerichtlichen Medizin. Berlin-Wien., 1919-1923.

4. JACKSON, CHEVALIER, and JACKSON, CHEVALIER L.: Diseases of the Air and Food Passages of Foreign Body Origin (Textbook). 1936.

5. MACCALLUM, WILLIAM GEORGE: A Textbook of Pathology. W. B. Saunders Co., 1936.

6. ASCHOFF, LUDWIG: Pathologische Anatomie. 8. Aufl. Jena, G. Fischer, 1936.

7. KAUFMANN, EDUARD: Lehrbuch der speziellen Pathologischen Anatomie. 9 and 10. Aufl., Berlin and Leipzig, 1931.

8. WEBSTER, RALPH WALDO: Legal Medicine and Toxology. W. B. Saunders, Philadelphia and London, 1930.

9. STRASSMANN, FRITZ: Lehrbuch der Gerichtlichen Medizin. F. Encke, Stuttgart, 1931.

10. SMITH, SIDNEY: Forensic Medicine. London, 1934.

667 Madison Avenue.

(a) — NEEDLE IN AORTA (WITH LANTERN SLIDES). (b) — BILATERAL EMPYEMA (LEPTOTHRIX).*

DR. DAVID H. JONES, New York.

A. W., age 28 years; Jan. 12, 1939; patient complained of dysphagia in the region of his epigastrium. Before entering the hospital, this pain was so severe that he visited a physian and was told that it was probably caused by gas. The dysphagia continued, and a routine X-ray examination made in the department store where he was employed showed a needle in the esophagus.

Jan. 18, 1939, he was referred to the Manhattan Eye, Ear and Throat Hospital but gave no history referable to swallowing a needle, although he did state that he often held pins in his mouth. The physical examination was negative.

Jan. 19, 1939, X-ray examination showed a long foreign body resembling a needle in the region of the esophagus at the level of the seventh rib posteriorly, lying at an angle of 45° in both anteroposterior and lateral views. The proximal end lay to the right on the anteroposterior, and anterior on the lateral view.

On the same day he was given preoperative medication consisting of nembutal, morphine, hyoscine and Forestier solution as a local anesthesia. He was then placed under a biplane fluoroscope but he was unco-operative, so that it was necessary to give him a general anesthesia. A 9 x 53 esophagoscope was passed, but the patient's position on the table was unsatisfactory, making the examination very difficult. Careful scrutiny of the esophagus revealed no foreign body and the biplane fluoroscope gave no assistance.

On Jan. 20, 1939, the patient was given a cotton ball saturated with barium and fluoroscoped as he swallowed. This was done by the resident physician. The cotton ball stopped suddenly at a point corresponding with what was supposed to

*Read at the meeting of the New York Academy of Medicine, Section on Otolaryngology, May 3, 1939.

Editor's Note: This ms. received in Laryngoscope Office and accepted for publication, June 15, 1939.

be the position of the needle. A feeding tube was placed *in situ* and an X-ray taken, which showed that the foreign body had apparently pierced the esophagus for a distance of approximately 1 mm., the remainder of the foreign body extending into the mediastinum.

Under general anesthesia, with the patient lying on a regular operating table, a 7 x 40 esophagoscope was passed and the esophagus was searched very carefully, both on introduction and on retrograde. This was confirmed by another operator. Laryngoscopy and bronchoscopy were done, and the right bronchus searched, as well as the middle and lower opening, but nothing was seen or felt by the applicator. The patient was returned to the ward very cyanotic, with barely perceptible pulse and shallow respirations. He was thought to be in shock. At 6 P.M., his condition was unimproved after receiving hypodermoclysis, heart stimulants, oxygen, carbon dioxide and external heat. At 9 P.M., his condition was still unimproved. The temperature was 102°, the pulse weak and uncountable, and the heart sounds weak. There was no evidence of cardiac dilatation by percussion. The patient was conscious and rational.

Jan. 21, 1939, at 7:45 A.M., his condition was the same, and at 8:20 A.M. the patient ceased to breathe.

POSTMORTEM.

When removing the anterior chest wall, dark blood could be seen through the pericardium, and when this was opened it was found to contain approximately 500 cc. of blood and clots, completely tamponing the heart.

The viscera of the thoracic cavity and neck, from and including the tongue down to the diaphragm, were removed *in toto*. The esophagus was opened from the cricopharyngeus to the cardia, and at a point $1\frac{1}{2}$ inches below the bifurcation of the trachea, the sharp end of the needle was found in the anterolateral wall, 0.5 cm. of it visible and pointing downward. The perforation in the esophagus through which the needle extended was about one-eighth inch in diameter, causing erosion of the edges of the perforation. The medical examiner felt that the needle had been in place longer than 60 hours. The eye of the needle extended through the anterior

and posterior wall of the aorta, about three-fourths inch above the free margins of the aotoric valves, and then through the posterior wall of the right auricle, with three to four puncture wounds of the anterior wall of the right auricle. On the inner surface of the posterior wall of the aorta there was a subserosal hemorrhage, 2 x 1 inch in size, and a thrombus at the site of the perforation, approximately the size of the little finger. The needle was an ordinary sewing machine needle, rusty in spots and possessing a very small eye; it was 1¼ to 1½ inches in length.

No gross evidence of a mediastinitis was present. There was no evidence of trauma in the larynx, trachea or bronchi, and the only trauma in the esophagus was at a point 1 inch cephalad to the needle, which consisted of an area of denuded mucous membrane, 1 x 2 cm. There was absolutely no evidence of perforation of the esophagus or bronchial tree after careful scrutiny by the medical examiner.

Diagnosis: Hemopericardium. *Cause:* Puncture wound (needle) of aorta and right auricle.

BILATERAL EMPYEMA (LEPTOTHRIX).

C. St. J., age 2½ years, white girl, in November, 1938, drank an unknown quantity of lye. She was taken to a hospital but she developed pneumonia and later measles, so little was done except a blood transfusion. When admitted to the Manhattan Eye, Ear and Throat Hospital, on March 11, 1939, she was badly dehydrated, was unable to take fluids, the milk which was given being returned; the weight was 18 pounds, and the subcutaneous tissue sparse. At esophagoscopy, on March 14, the 4 mm. bronchoscope reached a decided narrowing 16 cm. from the upper teeth; through it a No. 12 Levine feeding tube was inserted, subsequent X-ray showing it to be in the stomach. Thereafter considerable nourishment was retained daily, consisting of milk, karo, cereal, orange juice, beef juice, and the amount was increased daily. After a week a 200 cc. blood transfusion was given. The temperature remained nearly normal.

On March 24, a new feeding tube was inserted through the other nostril, but an X-ray showed it not to be in proper position; therefore, two days later another feeding tube was

inserted, following which her condition became poor, abdominal distention and respiratory difficulty appearing. An X-ray of March 27 showed the tube properly in the stomach, but there was increased density of the left lower lobe, suggestive of pneumonia, and an X-ray on the next day showed the density increased. At this time she was seen by Dr. Langman, who started sulfapyridine treatment. Neoprontosil was substituted for sulfapyridine the next day. Her temperature meanwhile rose to 103°, and a diarrhea appeared. Chest X-rays still showed increased density on the left side. An X-ray on March 30 showed the appearance of fluid or consolidation on both sides of the chest. Some food was regurgitated, and there was diarrhea. On March 31, neoprontosil was stopped. Nourishment had consisted of boiled milk and barley water.

On April 1, 100 cc. of a pink purulent fluid was aspirated from the left chest, and in it staphylococcus aureus and occasional pneumococci were found. Respirations improved temporarily, but on the next day she was more cyanotic and considerable tympanitis existed. April 3, she remained cyanosed, refused feedings, regurgitated food, and that day pulled out the feeding tube. It had become eroded and roughened. Fluid aspirated on that date from the left chest contained a leptothrix. Oxygen therapy was begun, April 3, immediately after a left rib resection by Dr. Wingebach. The pleural cavity was not opened.

April 4, 40 cc. more of thick pus was aspirated from the left chest, containing leptothrix. Dr. Wingebach advised letting in air and irrigation of the cavity with Lugol's solution; about 200 cc. of purulent fluid came out of the left side of the chest; a blood transfusion of 160 cc. was given. X-rays two days later showed marked effusion over the bases of both lungs, with a fluid level for each. Fluid removed from each side of the chest, April 6 and 7, was comparable with gastric contents, and grew leptothrix. The aspirations of the right chest were done, using constant suction until the needle was out. Fluids were given by hypodermoclysis as she took little by mouth, though some was given by rectum. In the needle track through the right side of the chest, an abscess of the chest wall developed and broke through a small opening in the skin after two days. On April 10, by X-ray, both pleural

cavities were half-filled by fluid; X-rays after chest aspiration showed the left side practically clear, some fluid remaining at the right base. Another transfusion of 240 cc. of blood was given. The left lung showed some re-expansion. From time to time she took a little milk by mouth.

On April 17, esophagoscopy under biplane fluoroscopy was done. The tube was replaced in the esophagus with difficulty, persisting in going to the right. Afterwards barium was put through this feeding tube, X-ray showing that it entered the right pleural cavity. Some of the barium was aspirated out through the feeding tube. Food given by the tube on April 19 was seen to flow out of the opening in the left chest. Dr. King noted that the right and left pleural cavities communicated. The next day the dressings on both sides of the chest were soaked by milk. With the tube in place the cavity was irrigated repeatedly with saline until the returns were clear. Then, keeping a constant suction through the feeding tube, this tube was slowly pulled up, nothing being obtained until at about 1¾ inches removal there was a sudden gush of pus, about 30 cc., but no more the rest of the way out. On the following morning, 25 cc. of pus was aspirated from the right pleural cavity.

On April 21, under local anesthesia, a gastrotomy was done and through it a feeding tube passed easily up the esophagus from below. The peritoneal cavity seemed normal and during the operation there was no evidence of a direct opening from the stomach into the pleural cavity. By this tube fluids were given after operation, none seeming to pass into the pleural cavities. Ten cubic centimetres of pus was aspirated from the right side of the chest, nothing being obtained from the left. There was no abdominal distention at any time after the operation, and the abdominal dressings remained dry. On the twenty-fourth, she began to have frequent stools, X-rays showed a beginning pneumothorax on both sides, cyanosis developed and the general condition grew decidedly worse.

On April 25, she was cyanotic and had laboring respiration before the dressing began. When the right dressing was removed, several cubic centimetres of pus flowed out of the needle hole; to allow postural drainage of pus out of the right pleural cavity, the child was laid on her right side. Thereupon, marked cyanosis and gasping respirations occurred.

Finger pressure was immediately put over the wound to prevent further pneumothorax, the color and respiration then improving. A tight pressure dressing was applied to the right side, excluding air from entering that side. Air could still be heard going in and out of the left side, however, and it was thought urgent to put a similar tight dressing on that side and then inflate the lungs by positive endotracheal pressure. Before this could be accomplished, respirations ceased and mouth-to-mouth insufflation of air into the lungs was carried out for four or five minutes to overcome the pneumothorax, but heart beats did not resume.

AUTOPSY FINDINGS (15 HOURS POSTMORTEM).

Gross Anatomical Diagnoses:

Generalized fibrinopurulent peritonitis.

Adhesive pleuritis, right and left.

Posterior mediastinitis.

Erosion of esophageal mucosa at cardia.

Absence of esophageal mucosa for a distance of 1¼ inches in the lower third, about opposite the eighth and ninth thoracic vertebrae.

Old perforation of the esophagus on right side, level of D8 vertebra.

Excised tenth left rib in posterior axillary line, with opening into the pleural cavity.

Perforation of right chest wall communicating with right costophrenic sulcus.

Hepatic consolidation of right and left lower lobes of lungs.

Passive congestion of kidneys.

Healing surgical incision of abdomen (epigastrium).

Healing surgical incision of anterior wall of stomach.

Generalized emaciation.

Pericardial effusion.

Specimens in Formalin for Microscopic Examination:

Left kidney.

Pancreas.

Right lower lobe of lung with attached pleura.

Left lower lobe of lung with attached pleura.

Right ventricular wall of the heart.

Gross specimens of esophagus and stomach, upper end of esophagus cut at level of the cricoid cartilage.

Upon opening the abdomen, the incision of the wall was found firmly closed and healing. The peritoneal cavity was everywhere filled with thin green pus, in which were flakes of pus and fibrin. Omentum was adherent to the anterior wall of the stomach. There was very little distention of the bowel. The surgical incision in the anterior stomach wall was tightly closed and nothing could be forced out of it from within by manipulation; one or two tiny beads of pus oozed out from about one of the stitches. The nasal feeding tube had been removed before the autopsy. The right diaphragm was at the fourth rib anteriorly, the left diaphragm at the fifth rib. There was no adhesion between the stomach or liver and diaphragm.

Before opening the chest, its wall was immersed in water and openings made under water into the pleural cavities; no air bubbled out. Upon opening the chest, no pneumothorax was present, the upper lobes of both lungs being fully inflated by air. The bases of the lungs were densely adherent to the diaphragm on both sides of the chest and to the chest wall save in the right costophrenic sulcus. The parietal pleura was 2 or 3 mm. in thickness throughout its lower half. An opening 8 mm. in diameter through the right chest wall in the axillary line communicated freely with the pleural space in the right costophrenic sulcus, but there was no opening into the diaphragm. On the left side, the lung, below and up to the opening of the rib resection, was very adherent to the lateral chest wall. Above that level was a large pleural space lined by thickened visceral and parietal pleura. There was no fluid in the pleural spaces.

On opening the pericardial sac a serous yellow fluid was found, about 30 cc. The heart had stopped in systole. There was no evidence of air or of air mixed with blood when the right ventricle and the pulmonary artery were opened *in situ*. No embolus or thrombus was present in the heart cavities or greater vessels.

The lungs were freed with difficulty and tearing because of dense adhesions to diaphragm, parietal pleura and mediastinum; they were cut off at each hilus. Upon section, the lower lobes showed a heavy red consolidation without froth or free fluid; the upper lobes appeared normal. The entire posterior mediastinum was involved in dense granulation tissue but there was no free pus or abscess cavity found. The esophagus was opened after its removal, together with the stomach. The upper two-thirds of the esophageal mucosa appeared normal; in the lower third, at a point corresponding to the level of the eighth thoracic vertebra, the esophageal mucosa was eroded entirely away for a distance of $1\frac{1}{4}$ inches of its length, and the edges of the right side of this erosion were granulated over to the outside of the esophagus; the left side, however, had been cut in the removal of the esophagus and it could not be determined whether or not there was an antemortem opening through the wall of the esophagus there. At the cardia there was an irregular absence of mucosa about 1 cm. in diameter. After the esophagus had been cut open, there did not appear to be any stricture or stenosis. The stomach lining was intact except for postmortem change in the mucosa.

No examination of the head or brain was made.

140 East 54th Street.

THE GENITONASAL AND GENITOAURAL RELATIONSHIPS.*†

DR. HECTOR MORTIMER, Montreal.

The experiments which Dr. Eggston has just reported are of great interest and I should like to thank him for the opportunity he has given me of studying and discussing his data with him today. Dr. Eggston has found evidence of an estrogenic effect upon the immature mouse uterus in extracts made from the nasal mucosa of the ox and, as Dr. Eggston has said, the possibility arises that the active material in such preparations may have originated in the nasal mucosa or may be present there secondarily.

THE GENITONASAL RELATIONSHIP.

Since the demonstration of the value of estrogenic substances in the control of atrophic rhinitis and ozena[1] and its confirmation,[2] the significance of such work as Dr. Eggston has just presented becomes of immediate interest to the otolaryngologist, and it may be well in this connection to review some of the known experimental facts that throw light on the nature of such reactions.

Man is not a good subject in which to study this question experimentally, but in other primates, such as baboons and monkeys, the experimental effects of estrogens on what are termed "sex-skins" are of fundamental authority. In these animals there is a periodic reddening and swelling of the skin of the perineum, of the nipple and of the face, chiefly around the eyes and upper parts of the cheeks, accompanying the sex cycle. Collings[3] (1926), Allen[4, 5, 6] (1927-1928), Morrell[7] (1930) and Parkes and Zuckermann[8] (1931) showed in Rhesus monkeys that this skin reaction could be produced experimentally by the administration of estrogen, and the last-named workers also obtained the reaction in a castrated female baboon. Bachman, *et al.*[9] (1935), studied the reaction of the skin to chronic

*Read at the meeting of the New York Academy of Medicine, Section on Otolaryngology, May 3, 1939.

†From the Department of Biochemistry, McGill University, Montreal.

Editor's Note: This ms. received in Laryngoscope Office and accepted for publication, June 15, 1939.

treatment with crystalline estriol in male and female Macacus monkeys and greatly extended our knowledge of the reactions which follow.

In 1936[10] we were able to show that the nasal mucosa of the monkey (Macaca mulatta) responds to the administration of estrogenic hormones in the manner of and synchronously with the changes known to occur in the nipple, sex-skin and face, and that this response is most clearly seen in the "middle" and inferior conchae which show reddening and/or swelling. It is a specific response and can be elicited in intact males as well as females, and in both immature and adult animals. It can also be produced in female castrates. The histological changes in the nasal mucosa are similar to those produced in the sex-skin.

In untreated immature and adult female animals, peaks of nasal activity periodically occur normally at intervals of about 28 days and these peaks are chiefly premenstrual in time; such naturally occurring cyclical activity in the nose of the monkey is most marked in the autumn months, when conception is most likely to occur. Fig. 1 presents graphically these cyclic changes in a normal adult female monkey; the top curve represents changes occurring in the face. The third curve from the top the changes occurring in the nipples, the fourth the changes in the skin of the back, and the fifth the changes in the perineum. The continuous lines indicate redness, and the dotted lines swelling. The second top curve indicates the changes in color of the middle and lower conchal mucosa. As in man, there is a good deal of normal variation in the color of the nasal mucosa and this is expressed in the chart as a zone which extends from "R" to "P" and includes variations from the healthy red of an animal in good condition to the pallid mucous membrane of a monkey in poor condition. Above this are recognizable three degrees of redness; "R3" is an intense brilliant red of a vividness almost that of red typewriter ribbon; "R2" and "R1" have also this quality, but less marked.

Fig. 2 shows the effect on the various sex-skins produced by administration of ketohydroxy estrin upon an adult female castrate monkey in which, when untreated, sex-skin activity is absent. The second curve from the top shows that this response is as active in the nasal mucosa as in the other areas

of specific response and, indeed, parallels them in degree and duration in a strikingly similar manner.

Histologically, Fig. 3, a conchal mucosa, clinically described as "R3," shows changes chiefly in the connective tissue of the

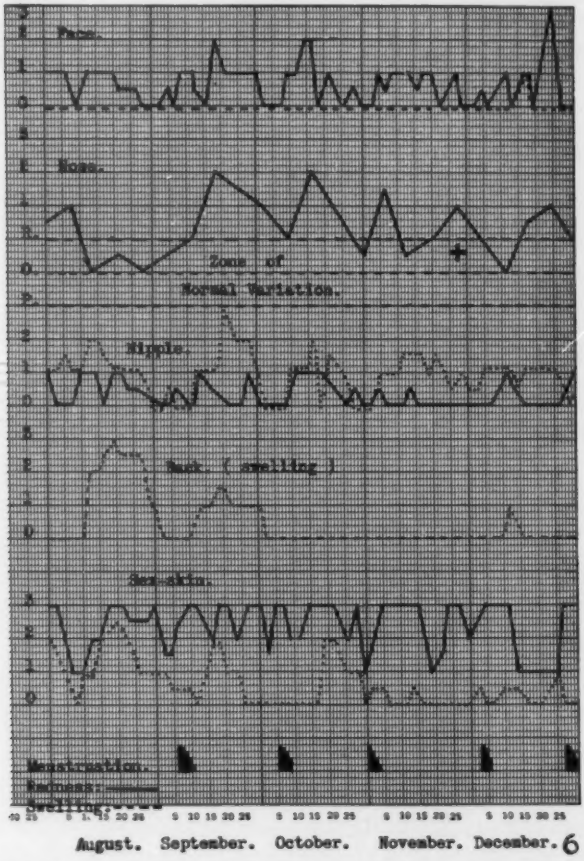


Fig. 1. Chart of periodic activity in the various "sex-skins" in a normal adult cyclic female monkey (Macaca mulatta). Untreated.

cavernous septa, where there is marked edema, spreading out the septal fibres, and the occurrence of young fibroblasts especially adjacent to the basement membrane, changes very sim-

ilar to those found in the corium of sex-skin of an animal subjected to this treatment.[9]

The mechanism of the production of such response to estrogen administration is of particular importance, and valuable

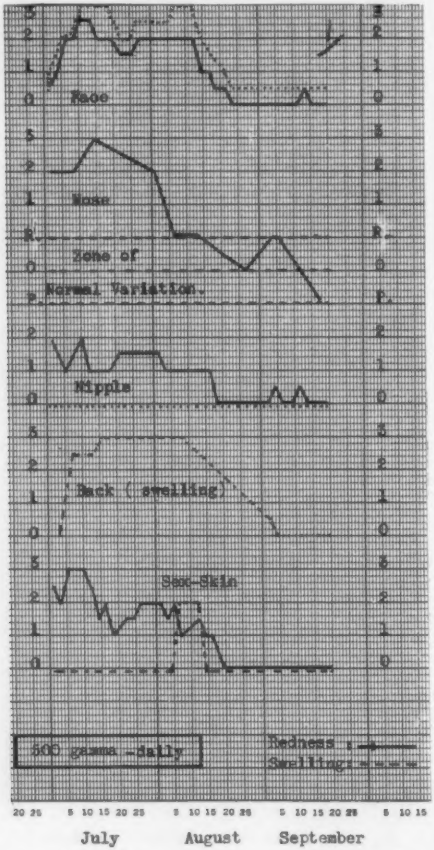


Fig. 2. The effect on the various "sex-skins" of treatment with ketohydroxy estrin in a castrate adult female monkey (Macaca mulatta).

light is thrown upon this by the work of Bachman, *et al.*[11] (1936). In a castrate adult female monkey, a segment of skin from a reactive area on the thigh was transplanted to

the anterior abdominal wall by means of a two-stage pedicle graft operation. After healing of the graft in its new situation, where the blood supply was that of a non-sex-skin and its previous segmental nerve supply was lost, on the administration of an estrogen the graft underwent a typical sex-skin reaction. The surrounding skin did not react. In another castrate female, the reverse of this operation was performed, a piece of non-sex-skin from the anterior abdominal wall

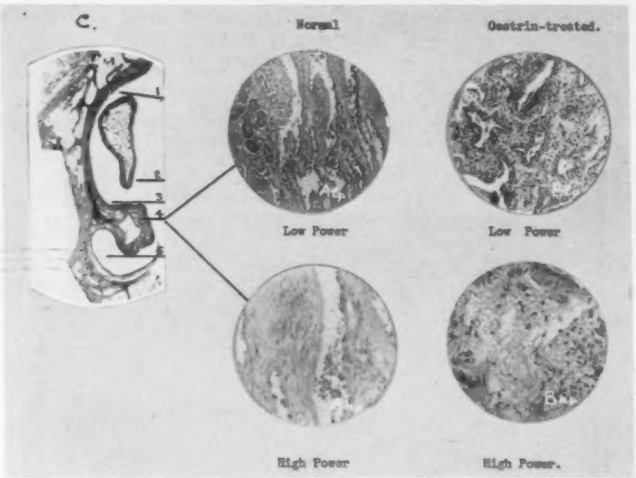


Fig. 3. "C" Coronal section, right nasal cavity in normal adult monkey. (1) Superior meatus; (2) "middle" concha; (3) middle meatus; (4) inferior concha — site of Sect. "A"; (5) inferior meatus.

Sect. "A." Normal structure of the cavernous tissue in the inferior concha of an adult female monkey (above: low power; below: high power).

Sect. "B." Inferior concha of adult female monkey after 14 days' injection of 1,000 gamma of crystalline ketohydroxy estrin in oil daily (above: low power; below: high power).

being transplanted to the genital area. Estrogenic treatment of this animal produced a typical color response in the genital area which, however, left the transplanted skin white and unchanged. The conclusion was drawn that these characteristic phenomena are probably determined by inherent peculiarities of the sex-skin itself.

Thus, in some primates certain cutaneous and epithelial areas have acquired in phylogeny the capacity of visible spe-

cific response to the ebb and flow of circulating estrin; in man, such visible response has been largely lost, although it still can be seen in the nasal conchae of the human female premenstrually and in pregnancy.[12] In Fig. 4 there is charted the progressive changes in human nasal mucosa in pregnancy and the urinary estrin in pregnancy (Browne and Venning,[13] 1936). This shows that at the end of the second month of pregnancy, the nasal mucosa is entirely normal in color and that the elimination of urinary estrin is less than 500 units per litre per day. By the end of the third month the nasal coloration is approaching the upper limit of normal. Between

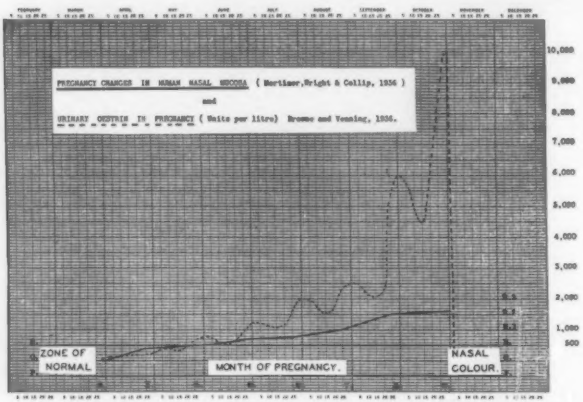


Fig. 4. Pregnancy changes in human nasal mucosa compared with urinary estrin output in pregnancy.

the fourth and fifth month of pregnancy the nasal color goes beyond the upper limit of the normal and from this time onwards the progressive increase in conchal reddening and swelling parallels the increasing elimination of estrin in the urine. After delivery, both fall off sharply.

That changes occurred in the nasal conchae during pregnancy is not a recent observation. Several observers noted this fact clinically at the end of the last century.[12]

The nasal mucosa in both men and women has also retained the capacity to respond specifically to estrin administered experimentally, and it is this which makes possible the control

of atrophic rhinitis in individuals in whom there is present a spontaneous atrophy of the mucosa and a depression of functional activity of its glands, which in turn make possible the secondary changes characteristic of ozena.

It has been suggested that tissues which have acquired phylogenetically the capacity of specific response to estrin may do so through selective concentration of "free" estrin at the reactive site, and experimental evidence to support this view was brought forward by Fisher, Krohn and Zuckermann[14] (1936), who found the presence of "free" estrin in the sex-skin and sex-skin exudate of monkeys, during the normally occurring cyclic skin reactions as well as in those resulting from estrin injection. It is on this basis that I would be inclined to account for the estrogenic effects that Dr. Eggston has shown to follow the injection of nasal mucosa extracts made from ox material. In short, I should be inclined to regard such evidence as supporting a genitonasal rather than a nasogenital relationship. It would seem that the former relationship is much better supported by experimental evidence than the latter, which is chiefly supported by the work of Koblanck and Roeder[15] (1912), Karpow[16] (1929) and Nemours[17] (1935).

THE GENITOAURAL RELATIONSHIP.

On first reporting the beneficial effect of intranasal administration of estrin in atrophic rhinitis and ozena (Mortimer, *et al.*,[1] 1936), we drew attention to the fact that we had at that time found six females and one male suffering from progressive deafness, as well as atrophic rhinitis. We suspected that these two constitutional disabilities might be related, at least to the extent of a common constitutional background, of which there was evidence. More recently (Mortimer, *et al.*,[18] 1939), further evidence to strengthen this view was found by us and in a total of 250 patients suffering from one or the other of the two diseases we found 42 cases in whom both defects were present. In a group of 35 cases of constitutional deafness we found that these two constitutional defects coexisted in the same individual and in the same familial stock more often than hitherto had been recognized. In such stock, the two defects might be distributed throughout one generation or in successive generations. We found that a treatment which acts specifically upon the nasal disease was capable when used

empirically of producing not only marked improvement in the aural defect in certain individual cases, but also a statistically significant amelioration in the hearing level of the group of constitutionally deaf as a whole.

In that report it was freely admitted that the experimental conditions were far from satisfactory during the period of investigation covered by the report. Steps have been taken to rectify these defective conditions and have been fully reported elsewhere (Burr and Mortimer,[19] 1939).

Since then, patients before and during treatment are examined at monthly intervals by means of a Western Electric 2A Audiometer in a soundproof room. They are also examined in this room by means of a "precision" audiometer in an open sound field, the patient being seated at one metre distance from a loudspeaker, which replaces the earphones, and a constant reference level of intensity is used for all frequencies. This audiometer uses electrical apparatus similar to the 2A audiometer but has a much larger power capacity, higher precision and is of wider frequency range and scope. It generates either pure tones or a "warble tone." This is a tonal effect produced by varying the frequencies over a small range about the mean frequency, the time of the complete cycle of this variation being one-thirtieth of a second. This warble tone is more readily recognized by the subject than a pure tone, reduces fatigue and reduces the effect of standing waves in the sound field. To increase the reliability of the patient's response, a technique is used involving successive trials by stimuli selected at random in the neighborhood of the threshold, based on the method employed in the Bell Telephone Laboratories for measurement of loudness (Steinberg and Munson,[20] 1936). At any given frequency, a number of intensities, differing by an equal amount and covering a range of intensity within which the threshold is known to lie, are introduced to the subject the same number of times, a zero stimulus being presented once. The order of presentation is determined by chance. Some of the sounds are certainly audible, others inaudible. When the tone is presented, a lamp beneath the loudspeaker lights and the patient signals "yes" or "no" by the corresponding push button. As already stated, the range covered by the trials of a given frequency extends from stimuli certainly heard to others certainly not heard, between which lies a point of complete uncertainty, and in

consequence the patient's replies to stimuli at this point will follow the laws of chance, and he will reply "yes" to 50 per cent of the times such an intensity is offered to him. This point is taken as the "threshold of hearing." In practice, it has been found adequate to present six intensities of a frequency, each presented five times, which with the zero stimulus makes 31 trials for testing a single frequency. Each of the seven frequencies throughout the hearing range is thus presented, and with three short rest periods it takes about three-fourths of an hour to investigate the patient's hearing by this method.

The trials of varying intensity at a given frequency are selected and recorded by what is known as "the random stimulus selector recorder." The patient's reply of "yes" or "no" is recorded by an electrically controlled typewriter and from this typewritten record a curve is drawn, the 50 per cent response point of which gives the threshold value.

This method of examination of the patient and of checking the data obtained by means of the 2A audiometer has been in use now for close on six months, and although it is not yet time for us to make a detailed report, we feel that we are justified in stating that in certain cases of those that have been treated and examined over an adequate period there has been evidence from both methods of examination of a considerable lowering of the threshold, which, we feel, can be accepted as real. I think one can venture to say that we have objective evidence that in certain cases estrin absorbed from the nasal cavities can produce a change in the acuity of hearing and that there is evidence of a genitoaural relationship in man.*

BIBLIOGRAPHY.

1. Mortimer, Hector; Wright, R. P., and Collip, J. B.: Atrophic Rhinitis; the Constitutional Factor and the Treatment with Estrogenic Hormones. *Canad. Med. Assn. Jour.*, 37:445-456, 1937.

2. Blaisdell, I. H.: The Use of Estrogenic Substances in Atrophic Rhinitis. The Laryngoscope, 48:699, 1938.

3. Collings, M. R.: A Study of the Cutaneous Reddening and Swelling About the Genitalia of Monkeys Macacus Rhesus. *Anat. Rec.*, 33:271, 1926.

4. Allen, E.: Menstrual Cycle of Monkeys Macacus Rhesus. Observations in Normal Animals of the Effects of Removal of the Ovaries and the Effects

*The four figures are reproduced by permission of the Canad. Med. Assn. Jour.

of Injections of Ovarin and Placental Extracts into the Spayed Animals. *Pub. Carnegie Inst., Wash.,* No. 380; *Contrib. to Embryol.,* 19:98:1, 1927.

5. ALLEN, E.: Reactions of Immature Monkeys Macacus Rhesus to Injections of Ovarian Hormone. *Jour. Morphol. and Physiol.,* 46:479, 1928.

6. ALLEN, E.: Further Experiments with an Ovarian Hormone on the Ovariectomized Adult Monkey Macacus Rhesus, Especially the Degenerative Phase of the Experimental Menstrual Cycle. *Amer. Jour. Anat.,* 42:467, 1928.

7. MORRELL, J. A.; POWERS, H. H., and VARLEY, J. R.: A New Source of the Ovarian Follicular Hormone. *Endocrinol.,* 14:28, 1930.

8. PARKES, A. S., and ZUCKERMANN, S.: Menstrual Cycle of Primates. Part II. Effect of Estrin on Baboons and Macaques. *Jour. Anat.,* 65:272, 1931.

9. BACHMAN, CARL; COLLIP, J. B., and SELYE, H.: The Effects of Prolonged Esterol Administration Upon the Sex-Skin of Macaca Mulatta. *Proc. Roy. Soc. Bull.,* 117:16, Feb., 1935.

10. MORTIMER, HECTOR; WRIGHT, R. P., and COLLIP, J. B.: The Effect of the Administration of Estrogenic Hormones on the Nasal Mucosa of the Monkey. *Canad. Med. Assn. Jour.,* 35:503-513, 1936.

11. BACHMAN, CARL; COLLIP, J. B., and SELYE, H.: Further Studies of Sex-Skin Reactions in Macaca Mulatta. *Proc. Soc. Exper. Biol. and Med.,* 33:549-551, 1936.

12. MORTIMER, HECTOR; WRIGHT, R. P., and COLLIP, J. B.: The Effect of Estrogenic Hormones on the Nasal Mucosa; Their Role in the Nasosexual Relationship and Their Significance in Clinical Rhinology. *Canad. Med. Assn. Jour.,* 35:615-621, 1936.

13. BROWNE, J. S. L., and VENNING, E. M.: The Assay of Urinary Estrin and Gonadotropic Substances During Pregnancy. *Amer. Jour. Physiol.,* 116:18, 1936.

14. FISHER, R. B.; KROHN, P. L., and ZUCKERMANN, S.: The Occurrence of an Estrogenic Substance in the Sexual Skin of Monkeys. *Biochem. Jour.,* 30:2219-2223, 1936.

15. KOBLANCK and ROEDER, H.: Animal Experiments on the Influence of the Sexual System by Nasal Interference. *Berl. klin. Wchnschr.,* 49:1893, 1912.

16. KARPOW, N.: Zur Frage des Zusammenhanges der Nase mit der Sexualsphäre. *Monatsschr. f. Ohrenheilk.,* 63:758, 1929.

17. NEMOURS, P. R.: Effect of Experimental Bilateral Turbinectomy on the Development of the Testes in the Rat. *Arch. Otolaryngol.,* 22:626, 1935.

18. MORTIMER, HECTOR; WRIGHT, R. P.; THOMSON, D. L., and COLLIP, J. B.: Intranasal Administration of Estrogenic Hormones in Constitutional Deafness. *Canad. Med. Assn. Jour.,* 40:17-22, 1939.

19. BURR, E. GODFREY, and MORTIMER, HECTOR: Improvements in Audiometry at the Montreal General Hospital. *Canad. Med. Assn. Jour.,* 40:22-27, 1939.

20. STEINBERG, J. C., and MUNSON, W. A.: Deviations in the Loudness Judgments of 100 People. *Jour. Acous. Soc. Amer.,* Oct., 1936.

McGill University.

CARCINOMA OF THE NASOPHARYNX WITH EXTENSION TO THE PETROUS PYRAMID. REPORT OF CASE.*†

DR. JOSEPH G. DRUSS, New York.

Carcinoma of the nasopharynx, because of the insidious nature of its growth and the sparsity of early manifestations, is frequently undiagnosed until late in the disease. Because of the relative inaccessibility and very high mortality rate, it is one of the most dread conditions with which the otolaryngologist is confronted.

Not infrequently, physical signs and symptoms, chiefly pain, tinnitus and impairment of hearing are related to the ear. Schlivek,[1] who reviewed, from the ophthalmologic standpoint, 38 such cases observed at Mt. Sinai Hospital, found signs referable to the ear in 14. New[1] observed aural symptoms in 29 of 79 patients studied, others in up to 50 per cent of the cases. It is important to emphasize the point that the aural symptoms are at times the first manifestation of the disease. This was true in four of the 35 cases reviewed by Needles.[2]

The following case is reported because of the interesting clinical problem in the differential diagnosis that it presented and because of the unusual histopathologic findings in the temporal bone.

Case Report: M. D., age 65 years, was admitted to the neurologic service of Dr. Strauss on May 30, 1936, with the following history: After an upper respiratory infection, six months prior to admission, the patient developed a right otitis media and mastoiditis, for which he was operated upon in another institution and from which he apparently recovered (pneumococcus types I and II). Four months ago, he developed pain in the left ear and, following a period of expectant treatment, a mastoidectomy was performed on that side in the same institution (pneumoccoccus type III). The patient was well until seven weeks ago, when the pain in the right ear recurred. These attacks of pain were intermittent, lasting only a few minutes at a time, and radiated to the temporal, frontal and occipital areas. For the past six weeks there was also blurring of the vision.

---

*Read at the meeting of the New York Academy of Medicine, Section on Otolaryngology, May 3, 1939.

†From the Otologic Service and Department of Laboratories of Mt. Sinai Hospital.

Editor's Note: This ms. received in Laryngoscope Office and accepted for publication, June 15, 1939.

Physical Examination: Both drums were flat and retracted, all landmarks were visible. The hearing was moderately impaired in each ear. In the routine examination by a member of the house staff, the nose and nasopharynx were found to be essentially negative. Percussion tenderness was elicited over the skull. A tender node on the right side of the neck was present. The right ankle jerk was greater than the left. Questionable left Babinski and Chaddock signs. The left pupil was larger than the right. Roentgen examination of the accessory nasal sinuses revealed clouding of both antrums, with evidence of thickening of the lining membrane of both sphenoids and of the left frontal. Roentgen examination of the petrous pyramids showed definite clouding of the right apex, with some decalcification of the ridge. Lumbar puncture revealed clear fluid, five cells, Pandy negative.

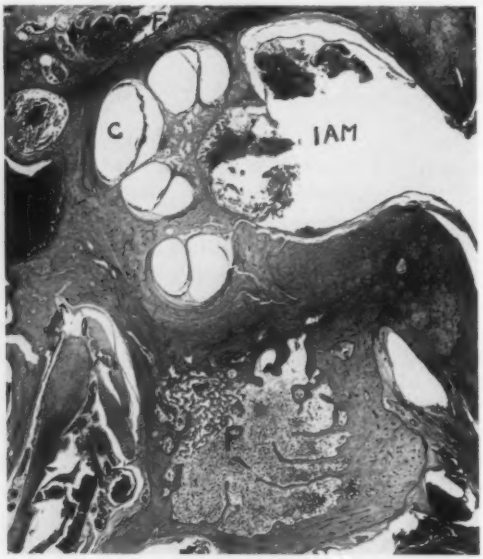


Fig. 1. Vertical section through the cochlea showing inflammatory reaction in the perilabyrinthine bony marrow (P). Granulation tissue, connective tissue and newly formed bone secondary to carcinoma in the apex of the petrosa (situated in sections more mesially than in that illustrated above). (C) represents cochlea, (F) facial nerve, (IAM) the internal auditory meatus.

Eight days after admission, the patient developed a right external rectus paresis and homonymous diplopia to the right. In view of these findings, the diagnosis of suppuration of the petrous pyramid was made secondary to extension from the mastoid. A modified Eagleton operation was performed. A perforation in the cortex of the superior surface of the pyramid was encountered about the level of the inner lip of the internal auditory meatus. Granulations were present on the adjacent dura. A series of pneumococcus type III vaccine was administered. The headaches subsided for a period of two weeks but then recurred with greater severity. The ptosis and VIth nerve palsy persisted.

Because of the persistent hemicranial pain, the question of re-exploring the petrosa was considered. Careful search, however, was made to rule out other possible causes. On Aug. 3, about two months after admission, an ulcerating mass was found in the nasopharynx, which on biopsy proved to be hornifying squamous cell carcinoma. The patient was discharged and referred to the radiotherapy department for treatment. The administration of three treatments of radium afforded him little relief of the headaches. Six days after discharge, he developed generalized convulsions and lapsed into coma. He was readmitted to the hospital with signs of acute meningitis, from which he ceased six days later.

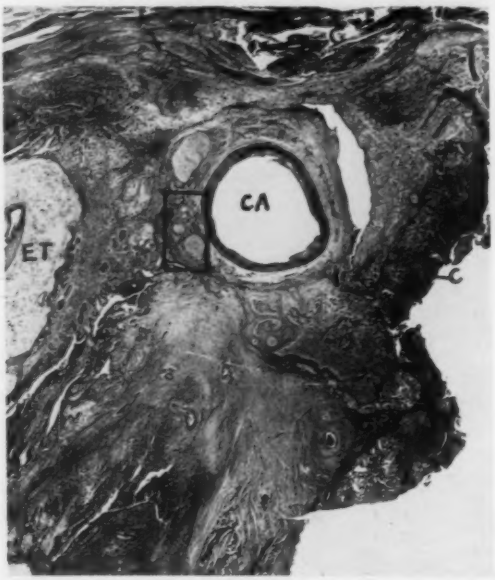


Fig. 2. Section through the apex of the petrosa. The carcinoma has destroyed to a great extent the bony structure, has infiltrated the adjacent dura, the pericarotid plexus of veins and nerves, the Vth nerve and its Gasserian ganglion, the cartilage of the Eustachian tube. (C) represents carcinoma, (CA) carotid artery, (ET) Eustachian tube, (G) Gasserian ganglion (V) Vth nerve.

Postmortem Examination: The basisphenoid shows an area of malignant invasion about one inch in diameter. The bone is completely absent at this site, with areas of extreme rarefaction about it. The apex of the right petrous pyramid is relatively soft and is infiltrated by this growth. The blood vessels of the dura in this region are matted down to the bone by the malignant extension. Purulent exudate is present over the base of the brain. Microscopic section of the brain shows evidence of meningitis.

Histologic Examination of the Right Temporal Bone: There is evidence of a previous mastoidectomy. The dura over the roof of the antrum is markedly thickened, infiltrated with cellular elements, and extends for

a distance into the antral cavity. It is necrotic in areas. The antral cavity is partially occupied by connective tissue. Purulent exudate is present in the tympanic cavity. The mucosa lining this cavity is swollen and is partially replaced by organized inflammatory exudate and fibrous tissue. These changes are particularly prevalent about the niche of the round window and about the oval window. Serous exudate is present in the perilymphatic spaces of the semicircular canal. There is an unusual amount of pigment in the modiolus and in the lamina ossea spiralis. There is a large bony dehiscence in the Fallopian canal as it courses

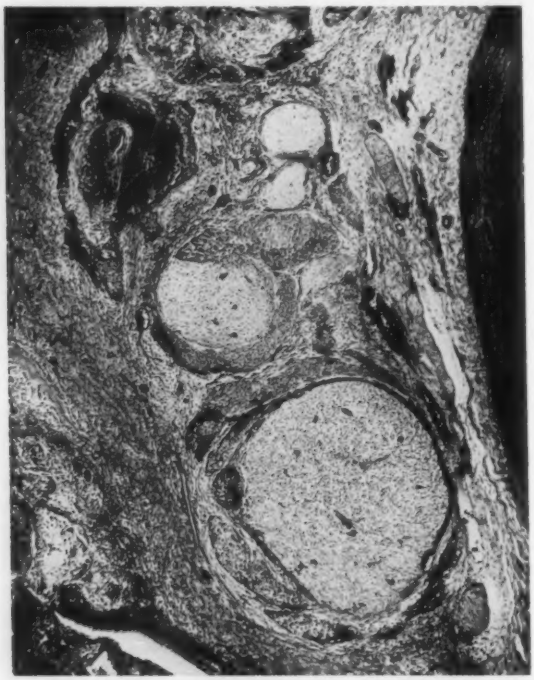


Fig. 3. Higher magnification of portion delineated in Fig. 2, showing carcinomatous infiltration of the perineural lymph spaces adjacent to the carotid artery.

through the tympanum, the facial nerve being lined at this site only by mucous membrane. The perilabyrinthine pneumatic and marrow spaces show considerable inflammatory reaction to the carcinoma, having been replaced by granulation tissue, connective tissue and newly formed bone (see Fig. 1). These inflammatory changes become more pronounced as the apex of the petrous pyramid is approached.

The apex of the petrous pyramid is infiltrated by squamous cell carcinoma, which has advanced by direct extension from the adjacent basi-

sphenoid. The neoplasm has invaded the bony structures of the petrosa, the dura of the middle and posterior fossae, the Gasserian ganglion, the Vth and VIth nerves and the pericarotid plexus of veins (see Fig. 2). The latter shows evidence of thrombosis. The perineural lymph spaces of the pericarotid plexus is likewise infiltrated by tumor cells (see Fig. 3). The carcinoma has eroded the cartilage of the Eustachian tube and has propagated along the deeper structures of the submucosa, but has not broken into the Eustachian cavity proper, the mucosa remaining intact. The tumor as it invaded the adjacent structures destroyed not only the old bone but also the newly formed bone laid down as a result of the secondary inflammatory reaction (see Fig. 4).

The carcinoma is of the stratified squamous type and is comprised chiefly of cells showing two phases in their development; one, the imma-

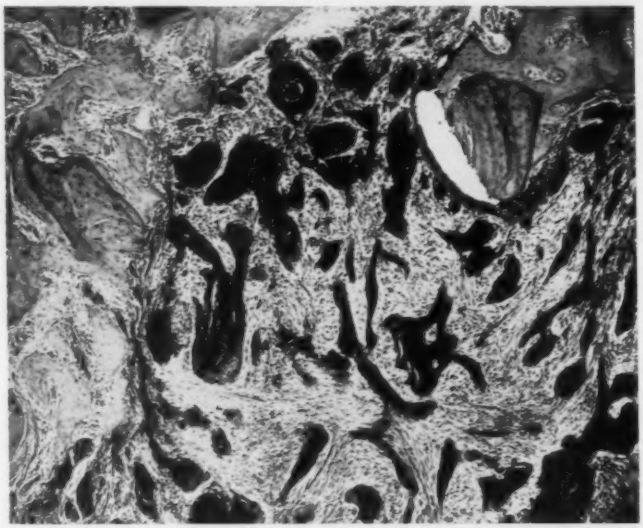


Fig. 4. The carcinoma is seen to invade the adjacent old and newly formed bone in petrous pyramid.

ture cell, which stains faintly, contains a single or multiple nuclei, is oval or stellate in shape and arborizes into various branches. The second phase is represented by the more mature cell, which takes on a heavier stain and shows strong tendencies toward hornification. These cells are arranged in ovals or circles, some exhibiting pearl formation, others undergoing degeneration.

COMMENT.

The most interesting feature in this case from the clinical standpoint is the difficulty in establishing the diagnosis of carcinoma of the nasopharynx. The patient having been oper-

ated for mastoiditis, developed three months later signs and symptoms of petrous pyramid involvement — *i.e.*, pain over the side of the face, blurring of vision, external rectus palsy, clouding and decalcification of bone in the petrosal apex on Roentgen examination and vague meningeal signs. The logical diagnosis of suppuration of the petrous pyramid was made. Upon exploration of this structure, a necrotic area in the bone was encountered. This procedure, however, afforded the patient little or no relief of symptoms. The underlying basis for these complaints was disclosed by re-examination of the nasopharynx two months after admission, when an ulcerating mass, which proved to be squamous cell carcinoma, was found. By this time the carcinoma had extended into the base of the sphenoid and the petrous pyramid. It is of importance to note that cursory examination of the nasopharynx made on admission revealed no evidence of tumor and that during this interval there were little or no signs referable to the nasopharynx. Salinger and Pearlman[4] and others have pointed out that one of the chief characteristics of this growth is its faculty of spreading widely beneath the mucous membrane so that ulceration or hemorrhage into the nasopharynx does not take place until late in the disease.

New[2] also emphasized the fact that there is a striking lack of nasal and nasopharyngeal signs in many of these cases. It should also be mentioned that not too infrequently unnecessary operations on various parts of the head are performed. To quote Needles, "These operations are directed toward the relief of one or more complaints and are undertaken without cognizance of the fundamental disturbance."

Histologic examination of the basisphenoid by Kramer and Som, and of the right temporal bone by the author, disclosed a widespread dissemination of the carcinoma in the nasopharynx to the adjacent bony structures, the nerves, blood vessels and overlying dura. The hemicranial headache and the external rectus palsy can be attributed to the direct extension of the tumor to the Vth and VIth nerves, respectively.

Of significance is the demonstration of carcinomatous infiltration in the perineural lymphatics, the latter serving as one of the routes of extension. These lymphatic spaces are normally contracted and usually cannot be identified as such. In the presence of infection or neoplasm, however, as in the case

herein described, they are observed as dilated spaces, infiltrated by purulent exudate or by tumor cells. While the carcinoma showed a strong tendency to erode the cartilage of the Eustachian tube and to spread widely in the deep structures of the submucosa, it made no attempt to ulcerate the adjacent mucosa. This is in accord with observations previously made by others.

That the inflammatory changes in the petrosa were secondary to the neoplasm and not to the previous mastoiditis is clearly demonstrated by the fact that the severity of the lesion diminished gradually as the more lateral perilabyrinthine regions were approached.

SUMMARY.

A case of carcinoma of the nasopharynx with extension to the petrous pyramid is presented, emphasis being placed on the difficult problem in diagnosis and on the histologic examination of the temporal bone. The importance of early and careful examination of the nasopharynx in all instances of unexplained hemicranial pain, aural symptoms or cervical adenopathy is again emphasized.

BIBLIOGRAPHY.

1. SCHLIVEK, K.: Ocular Manifestations of Malignant Nasopharyngeal Tumors. *Arch. Ophthalmol.*, 17:1055-1069, June, 1937.

2. NEW, G. B.: Syndrome of Malignant Tumors of the Nasopharynx. Report of 79 Cases. *Jour. A. M. A.*, 79:10-14, July, 1922.

3. NEEDLES, W.: Malignant Tumors of the Nasopharynx (with Special Reference to the Neurological Complications). *Jour. Nerv. and Ment. Dis.*, 86:4:373, 1937.

4. SALINGER, S., and PEARLMAN, S. J.: Malignant Tumors of the Epypharynx. *Trans. Acad. Ophthalmol.*, 40:281-316, 1935.

1040 Park Avenue.

MINNESOTA ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

SECTION ON OTOLARYNGOLOGY.

Meeting of May 31, 1939.

(Continued from March issue.)

Audiometers and Hearing Aids in Otologic Practice. Dr. Horace Newhart and Henry E. Hartig, Ph.D. (by invitation).

DISCUSSION.

DR. HARTIG: As Dr. Newhart said, we have been associated very pleasantly for several years in the investigation of hearing aids and audiometers, but occasionally he embarrasses me by ascribing to me in public authority which I cannot rightly claim. During the past year I have had the further pleasure of co-operating even more closely with the Department of Otolaryngology, in that I gave a course of lectures to graduate students in otology, covering the field of hearing aids and audiometers. Through this experience I have learned some things in which presumably otologists are interested and would like to know more. I should like this evening to keep the presentation informal and, in classroom fashion, I shall be glad to have you interrupt any time with questions.

Before me are a number of pieces of apparatus, some of which, though not new, many otologists have not seen in operation. First, the Western Electric Phonograph Audiometer, which is useful for screening large groups of school children. Though useful, it does not do a perfect job. It gives the person who is conducting the test information of a general kind. The child is permitted to listen to speech sounds of successively diminishing loudness and he writes down what he hears. You might think one could do that simply by taking a phonograph of the conventional type, putting on a special record and then have the children write what they hear; however, such a method would be very bad acoustically and would give no reliable information. The reason is that the sound intensity in different parts of the room would differ greatly. Consequently, children in the front row having defective hearing might correctly record the entire list of words, whereas a normal hearing child in a less favorable location would show an apparent hearing loss. To avoid this, individual phone receivers are provided, giving each child a direct acoustic path to the audiometer independent of room acoustics. (There followed a demonstration of the phonograph audiometer in which the output was amplified and impressed on a loud speaker for the audience to hear.)

Hearing tests may also be made with discrete pure tones, the familiar tuning forks being an example. Without question, in the hands of a careful operator and under favorable room conditions good results can be obtained; however, the tuning fork technique is relatively difficult. In point of simplicity, ease and accuracy of control, I do not see how it can compete with the modern electric audiometer. The latter also furnishes a pure tone, when properly constructed, and, moreover, its tone is sustained, not damped as is the tuning fork.

You may be interested to know how an audiometer works. Fundamentally, the principle is very simple and can be illustrated by means of the loud speaker microphone system which I have before me. By increasing the amplification beyond a certain point the loud speaker system sings or howls. (Here the demonstration was made.) A disturbance originating at the microphone is amplified by the loud speaker system and is again impressed on the microphone; when the amplified sound at the microphone is louder than the original disturb-

ance, it is again passed through the system and so on, ad infinitum, producing a sustained tone. Actually, of course, this oscillation is carried out in an electrical circuit without recourse to acoustic conversion, and in practice care must be taken if a truly pure tone is to be produced. Moreover, it is important that pure tones be used in measuring the threshold of hearing since the possibility exists that what the patient responds to at an audiometer setting of say 512 cycles per second is not the threshold loudness of the tone in question but harmonics of that tone, say 1,024 or 1,536, for which his hearing may be more acute. Consequently, it is important that an audiometer be held to certain minimum requirements, as has been done by the Committee on Physical Therapy of the American Association, in order to gain them acceptance. Not only must an acceptable audiometer tone be substantially free from harmonic content but the accuracy of the threshold intensity must fall within certain limits and, also, this setting should remain stable through the years. Also, the accuracy of pitch should be defined. Obviously, setting the standards for audiometers is a technical matter, into which we need not go further here; however, I should like to demonstrate the operation of two audiometers of different types: 1. the continuous sweep frequency, and 2. the discrete frequency audiometer. (Then followed a demonstration in which the output of each audiometer in turn was amplified and transmitted over the loud speaker system to the audience.)

Another important item in making hearing tests is the reduction of room noise to a sufficiently low level. The obvious way of doing this is to conduct the tests in a so-called soundproof booth. Constructing a satisfactory booth turns out to be, in the hands of a layman, a rather difficult thing to do. Consequently, we took up the matter of the construction of a satisfactory noise-reducing booth. The problem we set ourselves was to construct a booth, light, portable and sufficiently effective for the purpose. Contrary to what the words imply, one cannot construct a soundproof booth in the sense that the sound within the booth can be reduced to zero. One can only reduce the intensity of the sound inside in comparison with that on the outside. The booth which after much experimentation was evolved is shown in the slide. (Then were shown slides, photographs of the booth, diagrams of construction details, and curves showing the effectiveness in sound reduction at various frequencies. Also, there were shown slides illustrating diagrammatically the process of transmitting speech sounds from a speaker through a medium to a listener, and the effect of transmission losses either in the path of transmission or within the listener's conduction mechanism. A demonstration of speech sounds was made as they would be apprehended by a person having a specified hearing loss, as shown by an audiogram projected on the screen. Brief articulation tests were carried on, with the audience as judges, over the loud speaker system providing selective hearing losses.

Then the output of a carbon type hearing aid was amplified and the destructive effect of nonlinear distortion as produced in such a hearing aid was demonstrated by means of articulation tests. Finally, the output of a wearable vacuum tube type hearing aid was amplified and presented to the audience over the loud speaker system and the remarkable improvement in purity of speech transmission noted. It was emphasized, however, that the battery consumption of the vacuum tube hearing aids was considerably larger than for the carbon type.

DR. NEWHART continued the subject as follows: Ninety-five per cent of all of the hearing aids sold in the United States in 1937 were of the carbon microphone type. Until five years ago improvements in their construction and performance was slow, and the prejudice against their use was somewhat justified by their bulk, weight, conspicuousness and poor performance.

Their limitations included their small range; marked distortion with increased amplifications; abrupt peaking of the response curve in narrow bands, especially in the higher frequencies; a pickup limited to relatively short distances; deterioration of the carbon particles, causing reduction of output; disturbing internal noises from movement of the carbon particles and other causes, and

reduction or cutting off of the output with the microphone held in any position other than upright. Many of these defects have been overcome by certain manufacturers.

Recent improvements in carbon microphone aids include the amplifying microphone, the bone conduction receiver, more powerful midget receivers with individually fitted earpieces, and the use of better batteries, one manufacturer using two separate circuits to yield clearer speech reproduction.

The two outstanding advances in hearing devices are selective amplification and the production of wearable hearing aids with vacuum tube amplifiers and crystal receivers. By the selection of the component microphones, amplifiers and receivers from a number of such units, each possessing different characteristics, and the use of filters it is possible for the dealer to assemble a complete hearing aid, the performance of which most nearly meets the requirements of the individual as indicated by his audiogram. This is an important step in the right direction but it is not probable that hearing aids can ever be prescribed by the otologist with the same degree of accuracy as the oculist can prescribe glasses to correct refractive errors. Too many unpredictable variables exist, such as the variations in the individual's hearing acuity for the different speech tones; the instability of the output of electrical hearing aids, and the capacity of the ear to compensate for deficiencies at different frequencies make it unnecessary to prescribe a hearing aid on the basis of an audiogram showing hearing acuity at threshold levels at such small steps as octave letter intervals, as has been enthusiastically suggested by certain manufacturers. The ultimate test of the effectiveness of one hearing aid as compared with others is by the intelligibility test, with which every otologist should be familiar.

The vacuum tube hearing aid has certain advantages over the carbon microphone instrument, such as a wider range for frequency and intensity, ability to pick up sounds at greater distances and to give greater amplification without distortion. Persons whose hearing loss is so great that they cannot be benefited by a carbon type instrument can often be served satisfactorily with vacuum tube instruments.

With the vacuum tube device and a midget receiver having a well fitting, specially made earpiece, the wearer perceives sound by both air and bone conduction so effectively that the bone conduction receiver often is unnecessary except when perception by bone conduction is markedly greater than by air conduction.

The disadvantages of the vacuum tube type have been a relatively larger battery consumption, the susceptibility of the crystal microphone to the effects of heat and moisture, and the fact that when a single tube fails to function the instrument is useless until a factory replacement can be made. The carbon instrument usually gives timely warning of deterioration by a gradual diminution and irregularity of power and increased noisiness; however, the vacuum tube instrument is to be preferred for many patients who require extreme amplification with superior quality.

The rapid increase in the number of new hearing aids, each having its special features, has caused the prospective purchaser great confusion. This has imposed upon the interested, well informed otologist the important responsibility of helping his aurally handicapped patient to obtain the hearing device which will best meet his individual requirements. The more severe the hearing loss the more difficult is the problem. To adequately discharge this function requires some elementary knowledge concerning hearing aids and their limitations.

The prescribing or recommendation of a hearing device should be based upon a careful otological examination, including an audiogram for both bone and air conduction. With this help the otologist can determine if the patient will be served best by an air conduction receiver or a bone conduction receiver; whether he can get sufficient amplification with a carbon aid or requires the superior amplification afforded by a vacuum tube device.

Having determined the patient's requirements, it is best to advise him that he try out several aids of different makers whose products have proven acceptable to the Council on Physical Therapy. This precaution will best protect the purchaser, the otologist and the ethical manufacturer, and the patient will be better satisfied.

When any doubt exists as to the usability of the instrument prescribed, in fairness to the patient, a trial period on a reasonable rental basis should be arranged.

Occasionally, because of inexperience, the otologist, with the very best intentions toward the patient, mistakenly advises the purchase of an aid when the hearing deficiency is insufficient to warrant its use. In the past it has been found that persons having a loss of less than 35 to 40 dcb. in the better ear do not tolerate a carbon hearing device. With the better performance now afforded by improved models, the field of their application has been materially expanded. Persons in responsible positions, such as executives, teachers, stenographers and others, with a deficiency of less than 35 dcb. are today using modern devices with great comfort and satisfaction, being relieved from the anxiety and constant nervous strain of striving to hear without supplementary aid.

It is a serious mistake to advise a patient to postpone the purchase of a hearing device until he is forced to use one. Through such delay his hearing loss often becomes so great that when finally he gets his aid he is unable to interpret the distorted sounds he hears in terms of speech and becomes discouraged and unwilling to persist in the necessary effort to master this difficult situation.

All persons who can advantageously use a hearing aid should be advised to take up the intensive study of lip-reading or speech-reading to further supplement their defective hearing.

Every patient also should be informed that the purchase of a hearing aid does not afford the complete solution of his hearing problem. It is of the greatest importance that he make every effort to conserve his remaining hearing power by keeping in close touch with a personally interested physician or otologist who by watchful oversight of his health will correct or remove any discoverable conditions capable of causing further deterioration of his hearing organ.

The fact should be made known to physicians, educators and parents that many young children who have severe hearing impairments which prevent them from acquiring normal speech, by the early use of modern hearing devices in the home and local school, are able to acquire speech like normal hearing children and need not be sent to a special school for the deaf.

An important duty of the physician is to protect the hard-of-hearing public against imposition by certain unscrupulous high-pressure salesmen of inferior and often worthless hearing devices, who, especially in rural areas, victimize the unwary by exaggerated claims of the superiority of their devices. The public needs to be informed of the risk of buying hearing aids from persons other than those representing manufacturers of established reputation whose products preferably have been accepted by the Council on Physical Therapy of the American Medical Association.

Mr. L. A. Watson, of the Maico Co., was present and replied to several questions regarding audiometers and hearing aids from the manufacturer's point of view.

BOOK REVIEWS.

Diseases of the Ear, Nose and Throat. By Francis L. Lederer, B.Sc., M.D., F.A.C.S., Professor and Head of the Department of Laryngology, Rhinology and Otology, University of Illinois College of Medicine, Chicago; Chief of the Otolaryngological Service, Research and Educational Hospital. Second Revised Edition. Illustrated with 765 Halftone and Line Engravings on 463 Figures and 16 Full-Page Color Plates. Contains 840 pages with Index. Philadelphia: F. A. Davis Co. 1939. Price $10.00.

This very complete survey of the field of otorhinolaryngology has deservedly become one of the most popular reference books among medical students and post-graduates, and is now in its second edition.

The revised volume retains its original arrangement and now includes many new cuts and illustrations. The author has selected these with remarkably good judgment and has not hesitated to add or substitute for the benefit of clarity and teaching value. The photomicrographs of temporal bones by Crowe, and pathological sections, such as the plate on trichinosis of the larynx, are noteworthy additions.

There has been some rearrangement of material and alterations in the text following recommendations by Dr. Lederer's associates. The inadequate consideration of the subject of allergy and the author's failure to include his bibliography remain as our only exceptions.

This contribution is being received with enthusiasm both by teachers and students, and we highly recommend it to the specialist as well as the general medical practitioner. B. H. S.

Congenital Cleft Lip, Cleft Palate and Associated Nasal Deformities. By Harold Stearns Vaughan, M.D., D.D.S., F.A.C.S.; Professor of Clinical Surgery, New York Post-Graduate Medical School, Columbia University; Attending Surgeon, New York Post-Graduate Hospital; Consulting Oral Surgeon, Woman's Hospital; Consulting Surgeon, Southampton Hospital; Fellow of the American Association of Oral and Plastic Surgeons. With 259 Engravings. Two hundred ten pages including Index. Philadelphia: Lea and Febiger, 1940. Cloth, $4 net.

This volume is the "result of many years' clinical work and teaching in the care and surgical management of congenital cleft lip and palate," and its preparation has been stimulated by interested students who have been "unable to find the subject concisely covered in one treatise."

There is a good deal of the same material found in Dorrance's "The Operative Story of Cleft Palate," but the most emphasis is placed on the methods of repair that are in use at the present time.

The osteal uranoplasty is described as having bone flaps elevated from both sides in single clefts, whereas one of the main advocates of this procedure—

Davis—uses a bone flap from the cleft side and sutures it to a mucoperiosteal flap from the side attached to the vomer. The author, however, states that this "is not a procedure for the inexperienced palate surgeon."

One of the author's contributions is the use of thin lead strips placed all the way around the palate.

The section on lips has most of the standard procedures outlined with the author's method described of wiring the maxilla to bring widely separated alveoli together. The results of operations seem very good but the photographs are small and sometimes not very clear. The description of the Mirault operation is somewhat inaccurate, as it often is, because there are two Mirault procedures and neither of them has been very well illustrated originally.

The book is an excellent record of the author's work and should prove of great value. There are interesting contributions on prosthetic restorations, speech appliances and orthodontia by Dr. J. J. Fitz-Gibbons and Dr. L. J. Porter.

J. B. B.